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SPECIAL DENTAL PATHOLOGY

A WORK

ON

SPECIAL DENTAL PATHOLOGY

DEVOTED TO THE

DISEASES AND TREATMENT

OF THE

INVESTING TISSUES OF THE TEETH AND THE DENTAL PULP

INCLUDING THE SEQUELÆ OF THE DEATH OF THE PULP;
ALSO, SYSTEMIC EFFECTS OF MOUTH INFECTIONS,
ORAL PROPHYLAXIS AND MOUTH HYGIENE

518 ILLUSTRATIONS

BY

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PREFACE

THE writing of this book was begun five years ago, and its completion has been delayed in order that I might carry out long lines of experimental work upon several subjects which needed further investigation.

In the prosecution of the work, I have continually had the coöperation and advice of my sons, Dr. Carl E. Black, of Jacksonville, Illinois, and Dr. Arthur D. Black, of Chicago, which has been a very efficient aid.

My thanks are due the Research Institute of the National Dental Association for a five months' assignment of Dr. H. A. Potts, to assist me in carrying out investigations in my laboratory. I am under obligation to Dr. Thomas L. Gilmer, Dr. E. S. Willard, Dr. William Bebb, Dr. F. D. Leach, Dr. H. A. Potts, Dr. F. B. Noyes, and Dr. E. A. Schniedwind for suggestions and assistance. A number of the members of the classes of 1914 and 1915 of Northwestern University Dental School have aided, particularly in experiments in collecting deposits of salivary calculus and in testing the effects of various drugs upon the tissues.

The preparation of the illustrations and the final copy has been almost wholly under the management of Dr. Arthur D. Black, who has been in close touch with me constantly in this work. At my request, he has also written the article on Examinations of the Mouth, and Dr. Carl E. Black has supplied a compilation of the principal events leading to the development of antiseptic and aseptic surgery.

I am under special obligation to my daughter, Miss Clara Black, and to Mrs. Arthur D. Black, for valuable assistance in proofreading.

To all of these I wish to express my thanks.

G. V. BLACK.

Chicago, April 12, 1915.

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NOTE.— This table of contents has been prepared for use as an outline by students or practitioners who desire to make a careful synopsis in connection with the study of the subjects presented. All headings, subheadings and paragraph headings in the book are given in proper order, and the relations of these are shown by the positions of the headings in this table.

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Special Dental Pathology

INTRODUCTION

A STRICTLY dental disease is one that is peculiar to the teeth or their membranes, either in its causation, its nature, or in the tissues to which it is confined, and which can not occur elsewhere in the body. The tissues of the teeth are, in their histology and physiology, a distinct class. The membranes investing the teeth have peculiar histological and physical characters and forms suited to the functions of the teeth. These form a special assemblage of tissues, the pathology of which is unlike that of any other tissues of the body. It is this special pathology, together with the manipulation required in treatment, which has made dentistry a *specialty* in medicine.

I have previously written a book on the Pathology of the Hard Tissues of the Teeth, in which atrophy or hypoplasia, erosion, abrasion and caries were considered. In the present volume I shall include two principal groups: Diseases beginning in the gingivæ which may in their progress involve the peridental membrane and alveolar process, and diseases of the dental pulp and their sequelæ, including acute and chronic alveolar abscess, necrosis, etc. It will be found as we proceed that diseases of the peridental membrane occur as the result of either a preceding gingivitis, which first involves the peridental membrane at the gingival line of the tooth; or the death of the dental pulp, which first involves the peridental membrane at the apex of the root. From other than these two points of beginning, we have practically no disease of the peridental membrane, excepting as a result of some unusual traumatism. In both groups we are concerned with the investing tissues of the teeth, and this makes it especially advantageous to present a careful study of the physical functions of these tissues.

It is also advantageous to study the pathology of these two groups, as they include practically all of those foci in the mouth which endanger the general health. In the chronic suppurations of the peridental membrane beginning at the gingival line

and in many cases of chronic alveolar abscess, the investing tissues are detached from the cementum, and in the treatment of both we are confronted with the same problem of the impossibility of repair, due to the peculiar characteristics of the cementum. When such detachment occurs, the pus-soaked cementum becomes practically a dead tissue, which can not be exfoliated, and therefore maintains the chronic focus indefinitely.

Recent investigations of the relationship of these chronic foci to serious secondary lesions, demand the elimination of these foci, as well as the institution of more effective operating for their prevention in the future.

During practically the full period of my practice, I have carefully observed and recorded the pathological conditions of the peridental membrane, and for many years these diseases have been subject to special study. During the past few years I have devoted much time to their consideration, and it is for the purpose of giving my findings to the profession that this book is written. In fact, the publication of this book has been delayed in order to carry on recent investigations of the method of deposit of calculus, and other matters which were considered essential to a proper presentation.

A nomenclature sufficient for a satisfactory description and clear understanding of the various parts of the gingivæ and peridental membrane and their functions has been developed. Particular attention is given to the various groups of fibers and their functions in maintaining the teeth in position under normal conditions and in the movements which result from the cutting off of certain groups of fibers by disease. Likewise the changes which occur as a result of suppurative detachment, and the bearing which these changes have on reparative processes, are presented.

Diseases of the peridental membrane beginning at the gingival margins are perhaps, of all the diseases of the dental tissues, the least well understood. This is because of an insufficient knowledge of the histology, physical functions and special physiological relations and dependence upon each other, of the tissues involved, and the failure to study the local causes leading to the establishment of the disease by any efficient system of keeping records of cases in order to note their origin and progress. I know of no other group of diseases in which such a system of study is more necessary to a clear understanding.

There has been much confusion of ideas regarding the pathology of these diseases. This is largely because of the slow-

ness of their progress. We may see cases which have been progressing for twenty years, or even longer, before they have been regarded as serious, and afterward see the complete wreck of the denture. Any disease which progresses so slowly is especially difficult to study in its completeness. It is not like the study of, for instance, typhoid fever, in which practically the whole assemblage of phenomena occur within three or four weeks. One who sees many cases of such a disease comes soon to know the groupings of the various phenomena, and to know the physical manifestations of the disease in all its details. In the acute form of alveolar abscess the case may begin and run its course in from two to six days, and one easily gathers the essential symptoms, but in diseases of the peridental membrane, beginning at the gingival margin, in which the rise and progress usually extend over a number of years, the difficulties are greatly increased.

The different diseases of the gingivæ and peridental membrane, as well as the various causes, are separately considered, both as to pathology and treatment. It is of the utmost importance that these different conditions be recognized as a basis for correct diagnosis and proper treatment, although this seems not to have been done by the large majority of the profession. In medicine, an accurate diagnosis is the basis of successful treatment; it should be so in dentistry. The dentist who is able to make an exact and full diagnosis of the various diseases will have little difficulty in determining the best course to pursue in treatment.

The names or terms given to these conditions constitute one of the important features. These names are all very simple and in each instance both the cause and the tissue principally involved are included. Such a nomenclature is essential to a proper understanding. Inflammations involving the gingivæ only are definitely separated from those involving the peridental membrane, as a basis for rational preventive treatment of diseases of the peridental membrane, because gingivitis is a necessary antecedent of these diseases. The fact that deposits of salivary calculus destroy all of the investing tissues corresponding to the area of detachment from the root, generally without the formation of pus pockets, requires that the inflammation caused by deposits of salivary calculus be studied apart from all other inflammations of the investing tissues.

Studies of the deposit of salivary calculus have shown that the calcium element is brought into the mouth with the saliva in

the form of calco-globulin; also that the deposits are paroxysmal in character and of comparatively short duration at rather definite periods after meals. These studies indicate that the outpouring of calco-globulin results from digestion in excess of assimilation. Calco-globulin has been obtained from the saliva, also direct from Stenson's duct, and the specimens have been stained and photographed. Deposits occurring in the mouth, on traps constructed for the purpose, have been examined and photographed during all stages from the initial soft to the stony hard deposits. By a specially designed lathe for grinding microscopical sections of hard substances, the deposits of both salivary and serumal calculus upon the teeth have been studied and photomicrographs made which were not heretofore possible. These studies have indicated a thoroughly dependable system of treatment for prevention or control of the destructive inflammations resulting from deposits of salivary calculus.

In the study of the chronic suppurative detachments of the peridental membrane, in which pus pockets are formed, it will be shown that deposits of serumal calculus upon the cementum are never a primary cause of these pockets. Practically all cases may be accounted for as due to local causes, the treatment of which is usually simple, offering the key to effective prevention of this most destructive of mouth diseases, which is, of the mouth infections, the greatest menace to the general health.

In the consideration of the dental pulp, enough of the histology and physiological functions will be given to enable the reader to gain the best understanding of the diseases of this tissue. It will be noted that the classification of these diseases is based upon the clinical manifestations, rather than upon microscopical examinations which can not be satisfactorily applied in practice.

Radiography has enabled us to make much more accurate diagnoses of conditions within the maxillary bones than was possible previous to its use. The employment of the radiograph in the examination of cases of chronic suppurative pericementitis shows clearly the progressive absorption of the alveolar process subsequent to detachments of the peridental membrane from the cementum. The showing of cavities within the bone about the ends of roots, following pulp treatment in a very considerable percentage of cases, brings home the importance and absolute necessity for more careful technic and greater thoroughness in the handling of root canals. This should also

impress the need for more accurate diagnoses of pulp conditions and less of recklessness in pulp destruction.

This book is essentially a work on preventive treatment. For practically every pathological condition discussed, the possibilities and methods of prevention are presented. The special aim has been to point out the value in prevention of a closer study of the pathology, in order that careful observation and prompt recognition of the beginnings of these diseases will lead to better judgment and greater care in the finer details of manipulation in all operations performed. Effective prophylaxis against the diseases of the investing tissues can not result from the so-called oral prophylaxis treatments; this must be brought about by the practice of prophylactic dentistry, in which the effect of every operation in preventing or causing disease will be appreciated.

The place which the so-called oral prophylaxis treatment should occupy in practice will be stated. The necessity for the careful training of patients in mouth hygiene will be presented as an important element in the preventive and palliative treatment of most of the diseases considered. A separate chapter is devoted to the subject of mouth hygiene.

More rational medication than now generally practiced is strongly urged. This applies particularly to the use of caustics and antiseptics in the treatment of both the peridental membrane and dental pulp. The tendency of surgeons toward the abandonment of antiseptics in the treatment of wounds, on account of the effect of these in interfering with the activity of the tissues, should lead us to a similar course.

Conditions in the mouth are such that it is impossible to maintain asepsis. This fact, coupled with the fact that detachments of the peridental membrane from the cementum produce a constantly acting irritant, place these diseases in a class to themselves, entirely different from suppurations which occur elsewhere in the body. In treatment we should appreciate the exceptional powers of the mouth tissues in combating infections and should encourage them by maintaining the limit of cleanliness, rather than hinder them by the use of drugs which interfere with their activities.

In the consideration of so many closely related conditions, numerous duplications of statements occur. After a review of the completed text, it seems desirable, for the fullest understanding of each subject, that these repetitions remain.

Practically all of the illustrations are original. A considerable number are reproduced from my previous writings in the

American System of Dentistry and my own books and articles in dental journals. Others have been prepared especially for this book.

A lathe designed and constructed for the purpose of grinding microscopical sections of hard substances, such as teeth, deposits of calculus, etc., is illustrated and described in the appendix.

THE INVESTING TISSUES OF THE TEETH—GINGIVAE, PERIDENTAL MEMBRANE, CEMENTUM AND ALVEOLAR PROCESS

HISTOLOGY AND PHYSICAL FUNCTIONS

THE GUMS AND GINGIVAE

ILLUSTRATIONS: FIGURES 1-24.

THE gums clothe the alveolar processes and the hard palate, and the gingivæ invest the gingival portions of both the roots and crowns of the teeth. These divisions of tissue join each other by continuity without apparent demarcation at the crest of the alveolar process.* That is, there is nothing on the surface to indicate a change in the character or quality of the tissue. But at this point the soft tissue at once passes across between the adjoining teeth, through each interproximal space, and joins together the soft tissues covering the buccal and labial parts with the lingual parts, and surrounds each tooth. In doing this the teeth are completely invested with a *soft tissue alveolar process.* If this tissue were dissected away from the bony alveolar process and the teeth smoothly removed, it would consist of a considerable piece of tissue reaching around the arch, including the third molar on each side, through which there would be a hole (alveolus) corresponding to each tooth. While these divisions of tissue have much in common in their histological make-up, the gingivæ have tissue characters and functions not possessed by the gums. It therefore seems best to describe the characters common to both first, and then under the more specific definitions, to describe the gingivæ.

The gums consist of soft tissue noted for its compact inelastic firmness, which spreads from the crests of the alveolar

* While I have long considered the above the proper line of division between the gums and gingivæ, I have not heretofore had the courage to include so much tissue under the name of the gingivæ; but when I undertake to write a full description of these tissues, it seems absolutely necessary that the division be so made, because the crest of the alveolar process marks the logical boundary of the gingival covering of the teeth.

processes and covers the alveolar ridges well down and away from the teeth in all directions. Then a change in character to a soft mucous membrane occurs, which is reflected on the labial and buccal portions as the mucous membranes of the lips and cheeks from both the upper and lower arches. On the lingual side of the lower jaw it is much the same, the hard portions passing into the soft flexible mucous membrane of the floor of the mouth. In the upper jaw the dense membrane spreads over the entire palatal surface, back as far as the junction of the hard and soft palate. This hard inelastic tissue is known as the gums. Curiously enough, the plural form of the word is generally used, though the singular, *gum,* will be heard occasionally when the reference is to some particular spot. We also say *gum tissue,* and use the singular form in other such combinations.

The fibrous mat.

The basis of the gum tissue is a thick mat of inelastic fibers. Many of these fibers are large and are branched and connected in every direction in rather short lengths, forming a dense network, or mat. The periosteum, which is very firmly attached to the bone over this region, is also very closely interwoven with this fibrous network. In this union the two tissues retain their identity. That is, the periosteum retains its usual closely coherent form, and the fibrous mat of the gum tissue also retains its form, but the two are so united by interlocking of fibers as to prevent sliding movements of the one upon the other or upon the bone. This gives the parts their characteristic hardness and immobility.

One should have a clear understanding of the difference between such an immobile tissue and a very mobile tissue. If two fingers of one hand are placed on the back of the other hand crosswise, and if, while pressing firmly, the fingers are moved as far as the sliding of the skin will allow and the skin moved back and forth, it will be noticed that it will slide considerably. This will differ much in different individuals; in some it will move an inch or more, in others less. The so-called pulps of the palmar surfaces of the fingers are rather soft masses of tissue. If the pulps of the two middle fingers are placed together and moved upon each other with firm pressure, it will be noticed that this tissue, while soft and elastic, is comparatively immobile — much more immobile than the skin on the back of the hand. The gums are generally immobile. To demonstrate this, one may dry any part of the gums with a napkin and place the dry finger upon this

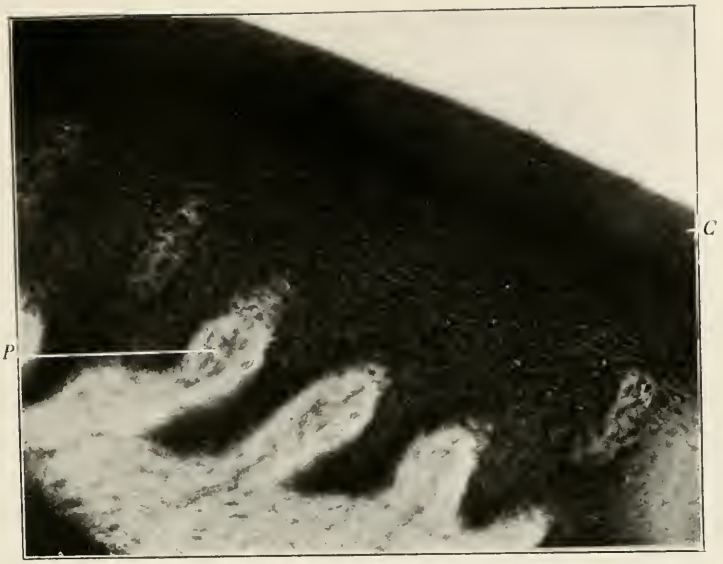

FIG. 1.

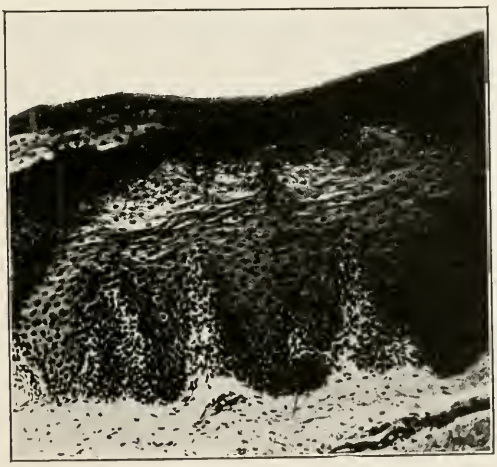

FIG. 2.

FIG. 1. Stratified squamous epithelium covering the alveolar process: C, Corneous layer. P, Papilla of connective tissue. *Noyes.*

FIG. 2. Stratified squamous epithelium from unattached mucous membrane of the mouth. The corneous layer is absent. *Noyes.*

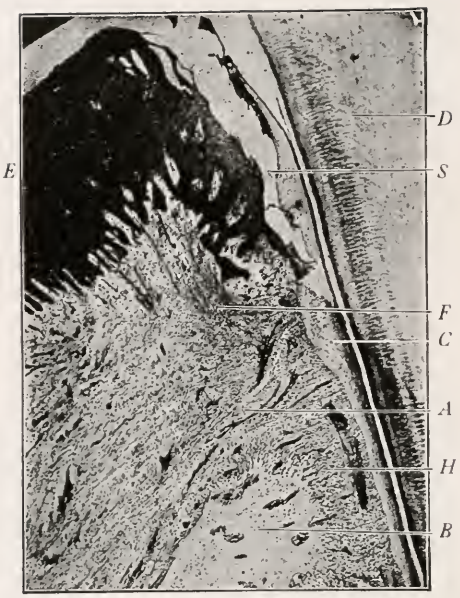

FIG. 3.

FIG. 3. Longitudinal section through the gingiva and the gingival portion of the peridental membrane. E, Epithelium. D, Dentin. C, Cementum. S, Subgingival space. F, Free gingivæ group of fibers. A, Alveolar crest group of fibers. H, Horizontal group of fibers. B, Bone of alveolar process. *Noyes.*

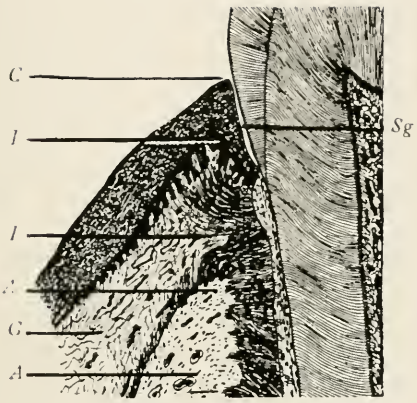

FIG. 4.

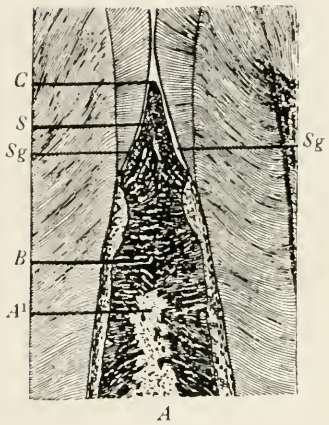

FIG. 5.

FIGS. 4 AND 5. Diagrams illustrating nomenclature of gingivæ.

FIG. 4. Bucco-lingual section through tooth and investing tissues. A, Alveolar process. A^1, Crest of alveolar process. G, Gum. B, Body of gingiva. F, Free gingiva. C, Crest of gingiva. sg, Subgingival space.

FIG. 5. Mesio-distal section through first and second bicuspids and septal tissue. A, Alveolar process. A^1, Crest of alveolar process. B, Body of gingiva. S, Septal gingiva. C, Crest of septal gingiva just below contact point. sg, sg, Subgingival spaces.

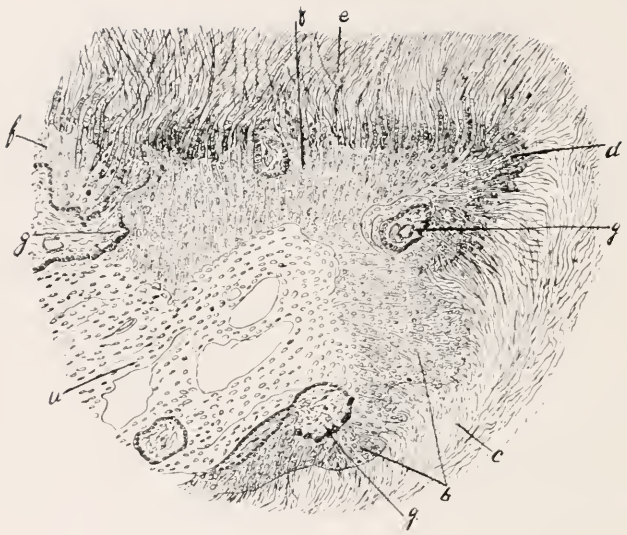

Fig. 6.

Fig. 6. Crest of the alveolar wall, from a perpendicular section. a, Haversian bone, which is left without stippling to render it more apparent. b, Subperiosteal bone, showing residual fibers. c, Periosteum. d, Extreme crest of the alveolar wall. e, Fibers of the peridental membrane. f, Bone formed by the osteoblasts of the peridental membrane. g, g, g, Points at which the absorption of bone is in progress.

spot, and try to move the tissue upon itself. One can not slide it at all. This explanation will give a full understanding of what is meant by the statement that the gum tissue is immobile. This quality of the tissue is so characteristic that it can not be missed in any examination of the regions named. These characteristics extend to the gingivæ in most of their parts, and may be said to be common to the two divisions of tissue.

Epithelium.

The whole of this region has a covering of strong pavement, or squamous epithelium. (See Figures 1, 2 and 3.) In most parts the tissues beneath are quite closely interdigitated into this epithelium, giving it a very firm hold, so that it is not easily scraped away. This epithelium has an especially strong growth and the surface cells are continually being shed away to give place to new superficial cells. These cast-off cells will be found liberally distributed in every specimen of the mixed saliva taken from the mouth.

Blood.

The blood vascular system, as everywhere in the mouth, is rich, and especially so in the capillary circulation. The blood vessels will be found everywhere in the tissue, winding in every direction among the interlacing white fibers. Accompanying the blood vessels, there are a smaller number of softer connective tissue fibers and cells filling in the interstices of the coarser fibrous mat.

Sensation.

The nerve supply is good, but the tissue is generally not very sensitive to painful impressions, though its sense of touch is fairly good. Its lack of sensitiveness to pain that would ordinarily be caused by the forcible movements of rough material over it is very striking when it is in normal condition. This is one of the curious phenomena of nature, stipulated and arranged for a purpose. This tissue, richly endowed with blood and nerves, lies on a surface where foreign substances are very frequently in contact with it, and yet as compared with most of the other tissues, it is markedly insensitive to these. The gingivæ have the same characteristics in all of their parts.

Why is it that this tissue is so insensitive to painful impressions? This is one of the very inconsistent things, at first thought, which becomes physiologically consistent when rightly

understood. These are defensive tissues, hard and rugged, not easily torn, bruised or lacerated, placed on either side of the dental arches and wound everywhere about the teeth. If these tissues, even though hard, were very sensitive, as their blood supply and their nerve supply would lead us to infer, we would be in pain from scrapings of many of the foods we chew and force harshly over their surfaces. Their hardness, which saves them from real injury, and their insensitiveness to pain, protects us from painful experiences in chewing food. This explains the physiological provision, and the necessity for it.

If we compare this insensitiveness of the membranes of the mouth with the extreme sensitiveness of the membranes of the eye, we will appreciate the differences in the endowments as to sensation given to different tissues to effect physiological purposes. The uses of the eye require the most perfect cleanliness of its surfaces. This is provided for by the lacrimal fluid poured over the surfaces to wash them, and the movements of the lids to prevent stagnation. The sensitiveness is so great that if the smallest mote sticks fast and will not move out, pain is promptly felt. This comparison should serve to impress the fact that the different tissues are differently endowed, as to sensation, and in many other ways, to effect physiological purposes.

Particular attention is called to the fact that this very insensitive tissue may be aroused into extreme sensitiveness (hypersensitiveness), especially by those conditions in which it is continually prodded and irritated. We often see this prominently manifested at points upon which a plate for artificial teeth binds too hard and is frequently being moved, or is working back and forth. Such points are particularly apt to become angry (if I may use the word in such a connection) and complain bitterly of every interference. We may also find very considerable sensitiveness aroused in cases of inflammation of the gums, and not very infrequently during the inflammatory process of the pointing of an alveolar abscess. Many other conditions will bring about similar results.

After all is said, we should still remember that under all ordinary conditions the principal function of the gum tissue is defensive. It is not very subject to diseases except as these spread to it, or are communicated by disease of contiguous parts. It is not a tissue which in general requires much attention from the dentist.

Healing powers.

In spite of the hardness and rigidity of the gum tissue, it will sometimes be lacerated in the crushing of hard substances over it. The rich blood and nerve supply is at hand to mend such breaks in the most speedy manner possible. Further, the nature of the tissue itself as to its inflexibility is such as to hold the parts in apposition instead of allowing them to spread apart, as most of the soft tissues are inclined to do when cut or torn. In this respect this tissue reminds one of soft vulcanizable rubber. If a knife cut is made in a bit of this soft rubber, the parts immediately return to apposition. This tendency, only somewhat less marked, will be found in the gum tissue, but has its greatest development in the gingivæ. This in itself contributes to very certain and rapid healing of small wounds.

In larger injuries, especially when the tissue has been torn away from the bones, we may find an opposite tendency — that is, to curl away. There are conditions of inflammation and swelling, which tend, for the time, to obliterate these characters. But even in the lancing of acute alveolar abscess, where the tissues are inflamed and swollen, and where considerable amounts of pus are discharged through the lips of the cut, I have seen the cut surfaces united within six hours. It is never safe to leave such a case after the evacuation of the pus, especially if more pus may be expected, without something in the wound to prevent it from closing too quickly.

The Gingivæ.

ILLUSTRATIONS: FIGURES 3-24.

The word gingiva, plural gingivæ, is derived from the Latin word gigno, which means to be born, to spring, to arise, and is applied to that portion of the gum tissue through which the tooth erupts, and later to that soft tissue immediately encircling the tooth.

That division of the tissues of the mouth which we call the gingivæ comprises the soft tissue which rests upon the crests of the alveolar process, including the crests of the septi which pass between the teeth, invests the gingival portions of the roots of the teeth and rises about the gingival portions of their crowns. (See Figure 3.) This division of the soft tissue is connected directly by the fibers of the peridental membrane to the roots of the teeth from the level of the crest of the alveolar process,

including the bony septi, to the gingival lines upon the teeth. At the crest of the alveolar process on the buccal, labial and lingual, the gingivæ join with the gum tissue without demarcation. They also rise about the teeth from the attachment at the gingival line, in a free border passing completely around each tooth and covering more or less of the gingival portion of the enamel surface of the crown. When in full and undisturbed health, this free border thins away to a knife-edge that lies very close against the surface of the enamel, but may readily be lifted from the enamel with a very thin flat instrument, disclosing the subgingival space between this free border and the tooth.

The large number of fibers of the character of those of the peridental membrane received into the gingival tissues from these several sources, and possibly others of like character and qualities developed in the tissue itself, serve to differentiate its histological characters from those of the general gum tissue. The coarser fibrous mat from the gum tissue continues into the gingivæ, but becomes finer and more closely woven and is reduced in proportional amount. While the general character of the tissue continues to be fibrous, there is in the gingivæ a larger proportion of cellular elements and blood vessels, and the tissue seems softer. Yet in any effort to move it or slide it upon itself, upon the bone, or upon the teeth, it is found to be practically immobile in any direction. It shows still more of the soft, rubber-like tendency to return its parts into apposition when cut, than has been mentioned in describing the gums. This very remarkable tendency facilitates the process of the healing of cuts and scratches of its surface in the most powerful way.

Among the tissues of the mouth, the gingivæ stand out in importance because of the fact that this tissue is the place of beginning of serious diseases, which are attracting more and more attention from the better men in dentistry, general medicine and surgery, and from many intelligent laymen as well. Up to the present time there has been surprisingly little study of these tissues in their healthy state, or of their functions or their physiological relations to the teeth and surrounding parts. What study has been given them, has been mostly of the empirical sort, or clinical studies that have not followed individual cases from early enough in their beginnings, nor long enough in their progress, to obtain the best results as studies of pathology.

For these reasons, every part of this tissue, as it appears in health, should be studied both anatomically and physiologically as closely as possible, with our present means. Even to-day,

any one who undertakes this study will find the literature very scant of facts bearing upon this particular subject. I shall be compelled to depend very largely upon my personal studies for what I may present.

PARTS OF THE GINGIVÆ.

The parts of the gingivæ are the *body,* the *free gingivæ* and the *septal gingivæ.* The body consists of that tissue which rests on the bony alveolar process, and forms a soft tissue extension of the alveolar process as far as the gingival line of the teeth. The free gingivæ and the septal gingivæ consist of that tissue which is grown upon the body, which encircles the gingival portion of the enamel of the crown of each tooth. Toward the occlusal, the free gingivæ thin down to a knife-edge margin, which I shall generally call the *crest* of the gingivæ. (See Figures 4 and 5.)

The free gingivæ may be conveniently divided into parts by naming the parts of the crown of the tooth against which they are imposed; viz., the *buccal gingivæ,* the *labial gingivæ,* the *lingual gingivæ.* Those portions which occupy the interproximal spaces are the *septal gingivæ.* The term *subgingival space* is given to the space between the free gingiva and the enamel which it covers.

THE BODY OF THE GINGIVÆ.

The body is attached to the gums by continuity of tissue on the labial, buccal and lingual sides of the teeth; and to the bony alveolar process by the fibers of its periosteum. It is attached by the fibers of the peridental membrane to the gingival portion of the roots of the teeth from the level of the crest of the bony alveolar process to the gingival line. This attachment to the root is on the average about two millimeters in width, encircling the root.

The fibers of the periosteum are short and their identity is quickly lost in the formation of a dense membrane, to which the superimposed tissue is united. (See Figure 6.) In any certain regions (except those of the actual attachment of tendons directly to the bones) in which strong attachments are to be made to the bones by attachment to the periosteum, the periosteum forms a membranous-like layer in that part of its thickness farthest from the bone, or in its outer layers, as opposed to the inner layers which lie upon the bone. Nearly all of the muscles which are attached directly without tendons, are united to such a layer of

the periosteum. I have found this form of the periosteum common about the crests of the alveolar processes. On the other hand, the fibers of the peridental membrane of this region are long and much in evidence in properly stained microscopic sections.

In these attachments the periosteum retains its character of close adhesion to the bone as described above, and unites suddenly, but very firmly, with the other tissue, so that there is not much spreading of fibers from it. The peridental membrane continues without change of form in the portions next to the cementum to the limits of its attachment at the gingival line. In this part of the peridental membrane the fibers are very plentiful, thick and strong. They radiate in part to the crest of the bony alveolar process, and in part to the soft tissues.

Groups of fibers in the gingivæ and peridental membrane.

The fibers of the peridental membrane form certain rather definite groups, in addition to which there are many scattering fibers. All of the groups, in both the gingivæ and peridental membrane, which deserve special description, will be mentioned here, in order that their relationship may be understood. Beginning with those fibers attached to the cementum at the gingival line of the tooth and progressing toward the apex of the root, we find the following groups:

The free gingivæ group, consisting of those fibers which pass out from the cementum near the gingival line of the tooth and then extend occlusally into the free gingivæ.

The trans-septal group, consisting of those fibers which pass across the interproximal space, connecting the proximal surfaces of the roots. Their attachment to the roots being between the gingival line and the level of the crest of the bony alveolar septum.

The alveolar crest group, consisting of those fibers which pass out into the body of the gingivæ and are attached to the crest of the bony alveolar process.

The horizontal group, consisting of those fibers which pass out at right angles to the long axis of the tooth and are attached to the bone of the alveolar process a little below the crest.

The oblique group, consisting of those fibers which pass from the cementum in an oblique direction occlusally, and are attached to the bone of the alveolar process. These oblique fibers constitute the body of the peridental membrane, or the fibers which cover the main body of the root portion of the tooth.

The apical group, consisting of those fibers which are attached about the apical portion of the root and extend in fan-shaped bundles to the surrounding alveolar process.

The free gingivæ group, the trans-septal group and the alveolar crest group extend within the gingivæ, while the horizontal group, the oblique group and the apical group are within the bony alveolus.

THE FREE GINGIVÆ GROUP. The fibers of this group extend outward for a short distance from the cementum, and then turn occlusally and are distributed to the free gingivæ. This group of fibers encircles the tooth completely, but is much thicker and stronger on the labial, or buccal, and lingual than on the proximal surfaces. As seen in longitudinal labio-lingual (or bucco-lingual) sections cut through the tooth and its investing tissues, it is a small, rather thick tuft of fibers turning toward the incisal (or occlusal), but if we consider the entire circumference of the tooth, the fibers of this group make up quite a mass of tissue, contributing to the rigidity of the gingivæ. It is the smallest of the groups of the gingival fibers of the peridental membrane. This group probably has a considerable influence in maintaining the free gingivæ in their positions of close adaptation to the teeth. (See Figures 3, 7 and 8.)

THE TRANS-SEPTAL GROUP. The fibers of this group arise from the proximal surfaces of the gingival portion of the roots of the teeth, and pass across and through the septal gingivæ over the bony septum from tooth to tooth, and from tooth to tooth, recurring in each interproximal space, attaching the teeth together continuously from one third molar around the arch to the third molar of the opposite side, in both upper and lower arches. In a good many instances this group of fibers is composed of a number of bands which pass irregularly across from tooth to tooth. These are sometimes intermingled in a plaited or interwoven form. In studying these, it seems that they are capable of making the pull as well as, or better than, those which pass directly from one tooth to another in a straight line. The effect of these fibers is to bind the teeth more firmly together in the mesio-distal direction and especially to hold the contacts of the teeth tight.

In histological sections which are cut horizontally through two or more teeth and their investing soft tissues, beginning with the crests of the free gingivæ and going rootwise as the sectioning proceeds, we come upon this group of fibers passing

from tooth to tooth, and always find it strongly expressed. It is composed of many strong fibers, even after scattering many others upward into the septal gingivæ. Often, in studying these, it has seemed to me that all of these fibers could not be attached to the cementum of the tooth, there are so many, and that some of them must arise within the tissue. These fibers have a work to do of physiological importance that has been mentioned, and it will shortly be discussed. (See Figures 8, 9 and 10.)

The alveolar crest group. The fibers of this group pass over to and are inserted into the crest of the alveolar process. This group has sometimes been called the dental ligament, though it has not the characters of a true ligament. These fibers appear to best advantage in longitudinal labio-lingual (or bucco-lingual) sections. In studying the structure in such sections it is often apparent that the periosteum covering the alveolar walls extends over the curve of the crest of the alveolar process and a short distance on the labial (or buccal) surface, to give place for the attachment of this group of fibers. This group forms a strong band of fibers completely encircling the tooth. It is, however, much stronger in its labial, buccal and lingual than in its proximal portions.

The function of this alveolar crest group of fibers is to assist the horizontal group in sustaining the tooth in its position in its alveolus, especially against lateral motions, and yet allow that slight motion necessary to the tooth in performing its function in mastication. This will be better understood after studying the *pull* and the balancing of the *pulls* of different groups of fibers, and the influences of disturbances brought about by interferences with this balance of the pull exerted by the various groups of fibers upon the teeth. (See Figures 3, 7 and 8.)

The free gingivæ.

The free gingivæ are soft tissue processes growing out from the body of the gingivæ and covering a portion of the enamel surface of the crowns of the teeth. They are on the labial, buccal and lingual surfaces of the teeth, and join the septal gingivæ at the angles of the teeth. This part of the tissue has no attachment to the teeth, after passing occlusally of the gingival line, but is simply closely fitted about them; hence the term *free gingivæ*. The height of the free gingivæ upon the teeth is variable, from one to five millimeters, sometimes even higher in children. In rising on the gingival portions of the crowns of the

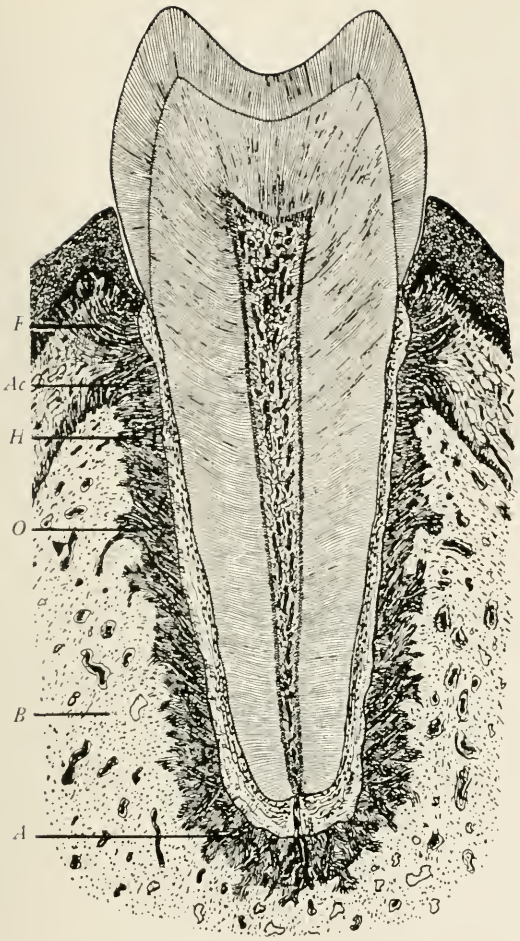

FIG. 7.

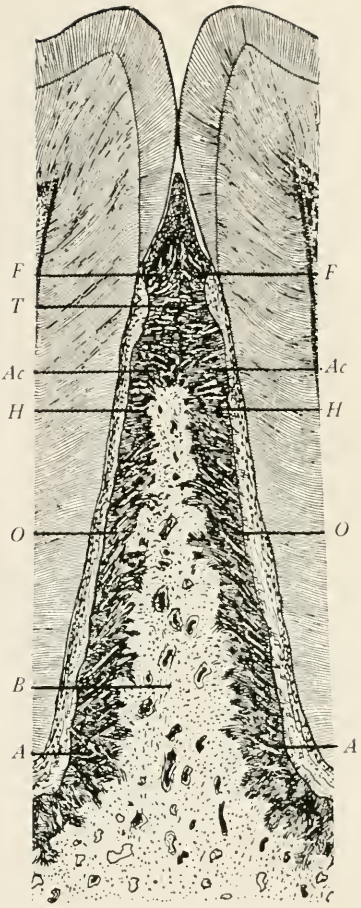

FIG. 8.

FIGS. 7 AND 8. Diagrams illustrating groups of fibers of the gingivæ and peridental membrane.

FIG. 7. Bucco-lingual section through a bicuspid tooth and investing tissue. F, Free gingivæ group of fibers. AC, Alveolar crest group of fibers. H, Horizontal group of fibers. O, Oblique group of fibers. A, Apical group of fibers. B, Bone of alveolar process.

FIG. 8. Mesio-distal section through two bicuspids and septal tissue. F, F, Free gingivæ groups of fibers into septal gingiva. T, Trans-septal group of fibers from tooth to tooth. AC, AC, Alveolar crest groups of fibers. H, H, Horizontal groups of fibers. O, O, Oblique groups of fibers. A, A, Apical groups of fibers. B, bony septum of alveolar process.

FIG. 9.

FIG. 10.

FIG. 9. Cross section of the central and lateral incisors a little to the incisal of the crest of the alveolar septum. a, Portion of central incisor. b, Lateral incisor. c, Pulp chamber of lateral incisor. d, d, Cementum of central incisor. e, e, Cementum of lateral. f, Trans-septal fibers of the peridental membrane extending from tooth to tooth continuously. These are attached in the cementum of each tooth. g, g, Fibers of the peridental membrane, which join with the coarse fibrous tissues, h, h, of the gingivæ. j, j, Epithelial covering of the gingivæ.

FIG. 10. A portion of the peridental membrane between two incisors of a young sheep, showing the trans-septal fibers extending from tooth to tooth. *Noyes.*

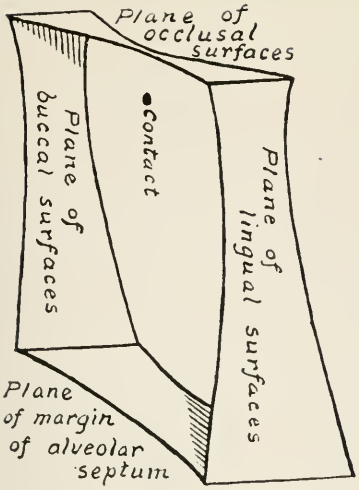

FIG. 11.

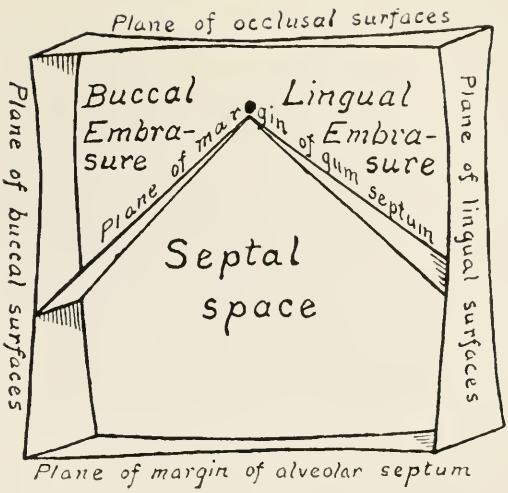

FIG. 12.

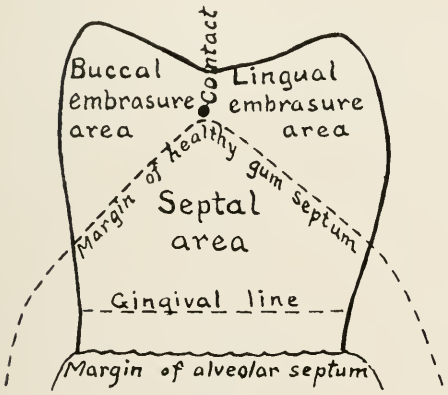

FIG. 13.

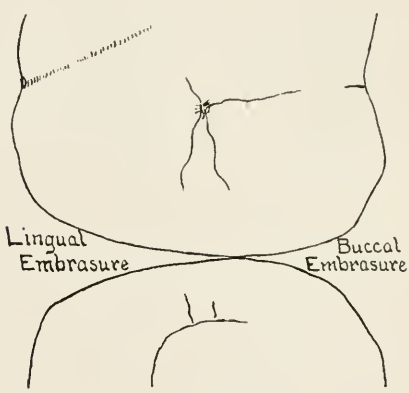

FIG. 14.

FIG. 11. Diagram to illustrate shape of interproximal space. If the rectangular frame is placed between two spheres which are in contact at the point indicated, the space within the frame and between the two spheres would be that of an interproximal space between the bicuspids and molars, which might be described as a rectangular section of a biconcave sphere.

FIG. 12. Diagram to illustrate the three divisions of the interproximal space. The buccal embrasure consists of that portion of the interproximal space to the buccal of the contact point which normally is not filled by the septal gingiva. The lingual embrasure consists of the corresponding portion of the interproximal space to the lingual of the contact point. The septal space consists of that portion of the interproximal space which is normally filled by the septal tissue. This space may be described as a pyramid set upon a rectangular solid.

FIG. 13. Diagram to illustrate the areas on the proximal surface of a tooth. The buccal embrasure area, the lingual embrasure area and the septal area are names given to those portions of the proximal surface of a tooth which correspond to the similar divisions of the interproximal space.

FIG. 14. Diagram to illustrate the relation of the embrasures to the point of contact. The portion of the interproximal space which is normally open (to the occlusal of the septal tissue), is divided by the point of contact into a buccal embrasure and a lingual embrasure.

FIG. 15.

FIG. 16.

FIG. 17.

FIG. 18.

FIG. 19.

FIG. 20.

FIG. 21.

FIG. 22.

FIG. 23.

FIG. 24.

FIGS. 15 TO 24. A selection of teeth to show their gingival lines. Figures 15 and 16, upper central incisors. Figure 17, an upper cuspid. Figures 18 and 19, mesial and distal views of an upper first bicuspid. Figure 20, an upper second bicuspid. Figure 21, an upper first molar. Figure 22, a lower incisor. Figure 23, a lower bicuspid. Figure 24, a lower first molar.

The curvature of the gingival line on the proximal surfaces of the incisors and cuspid may be compared with those of the bicuspids and molars.

teeth, the gingivæ thin away to a knife-edge margin. A thin blade may readily be passed between the gingiva and the enamel, raising the tissue and exposing the subgingival space between it and the enamel. This tissue has no attachment to the tooth until the gingival line on the tooth is reached, which means that the attachment is to the cementum only.

The form of the free gingivæ to a labial view of the incisors and cuspids is a smooth, graceful curve of about one-third of a circle with the concavity toward the crown or incisal edge of the tooth. This joins with the septal gingivæ at the angles of the teeth, continuing the curve into the embrasures well between the angles.

The septal gingivæ.

The septal gingivæ are processes of soft tissue growing up from each septum of the body of the gingivæ, and under normal conditions fill each interproximal space to its contact point. At the angles of each of the teeth the septal tissue joins smoothly with the free gingivæ by continuity and even fulness of form as the one passes imperceptibly into the other. The division between the two is a matter of form, and the naming of them separately is for convenience in description. But the difference in form is very real. Each septal gingiva fills a space between the proximal surfaces of two teeth. Each labial, buccal or lingual gingiva clings to and clothes an otherwise free surface of the enamel. Each septal gingiva has two subgingival spaces, a mesial and a distal, each lying against the surface of a tooth; while each labial, buccal or lingual gingiva has but one subgingival space. There is a difference in the firmness and plasticity of the septal gingivæ as compared with the labial, buccal or lingual gingivæ. This tissue, being broader and thicker, has within it much more of plastic tissue and is much more easily compressed than the free gingivæ. After a considerable compression, as by food impaction, it will again assume its normal form, provided the compression has not been too long maintained. The dangers which threaten the two parts of the tissues are also different, as we will find in the study of their pathology. For these reasons they should be named separately.

When normal and of good form, the septal gingivæ are more or less wedge-shaped and fill the greater portion of the space between the teeth from the gingival line to the contact point, the occlusal edge or surface sloping away from the contact

point buccally and lingually toward the gingival to the level of the buccal and lingual free gingivæ. The open spaces on either side of the contact point, to the occlusal of the septal tissue, are called the buccal and lingual embrasures. It is through these that food, crushed between the teeth and divided by the contact points, glides upon the sloping surfaces of the septal gingivæ to either side of the arch. This sliding is facilitated by the widening of the embrasures to the buccal and lingual, by the slope of the surfaces of the septal gingivæ and by the lubrication by mucus.

It should be particularly noted that this description provides a deeper filled portion and a superficial open portion of the general interproximal space; the deeper portion, occupied by the septal tissue, may be termed the *septal space,* while the superficial portion consists of the *buccal (or labial)* and *lingual embrasures.* (See Figures 11, 12, 13 and 14.)

The *form* of the surface of the septal gingiva which is exposed to the food which glides over it, is very similar to that of an inverted letter V, with the angle of meeting of the two legs of the letter at the contact point, and the legs extending buccally and lingually. The angle formed by the two lines may be a right angle in the incisor and bicuspid regions, or even less when the teeth have long bell-shaped crowns. Between thick-necked teeth, especially between the molars, it may be an obtuse angle, the slopes to either side being comparatively flat, yet there is sufficient slope to the buccal and lingual, aided by the broadening of the open embrasures and by the lubrication by mucus, for food separated by the contact to glide to the sides of the arch without difficulty. The best form of the gingivæ does not permit any unevenness in the fulness. Any depression — one that lowers a free margin and thickens it especially, is a fault which endangers the integrity of the tissues by facilitating lodgments on the teeth.

At the point of contact, between any two teeth, the septal tissue is reduced to a very thin edge; this becomes thicker progressively as we pass toward the gingival line, to fill the space made by the greater separation of the surfaces of the teeth, so that in some of these spaces the tissue becomes a comparatively thick mass. In the buccal and lingual directions, the thickness of the tissue increases as the teeth round away from each other in forming the embrasures toward their buccal and lingual surfaces, until it meets evenly with the free gingivæ at the angles of the teeth. In this slope, between the teeth, the surface of the

septal tissue is flat mesio-distally, and joins against the teeth in a right angle as it is reflected to form the surfaces of the subgingival spaces to the mesial and distal sides of its substance.

EPITHELIUM OF THE GINGIVÆ.

The epithelial covering of the gingivæ is one of the very important elements of their histological structure. Immediately after passing the position of the line of the crest of the bony alveolar process in going toward the crests of the free gingivæ, we find the epithelial covering becoming thicker and stronger, with the cellular elements themselves smaller and more closely interwoven. As the epithelium becomes thicker, the interdigitation of the connective tissues beneath, into the epithelial covering, becomes longer, more closely set and finer. In proportion as the epithelial covering becomes thicker, do we find the connective tissue elements becoming smaller, more thickly set and finer, until they are reduced to fine strings of connective tissue elements, each carrying a minute arteriole and returning vein with a rich little capillary circulation about them, thus carrying an arterial circulation almost completely through this thick mass of epithelium for its growth and support.

If the superficial epithelial cells in the live tissue in the gingivæ are scraped away with a sharp instrument, blood will ooze out long before any considerable portion of the epithelium has been removed. The bleeding is the result of cutting the ends of the long connective tissue prolongations into the body of the epithelium, which carry the blood for the support and quick growth of epithelial cells for the repair of abrasions and cuts received in chewing coarse foods. If a careful examination of this scraped spot is made on the morrow, no trace of the injury will be found. Each of these minute ends of connective tissue, carrying these delicate blood vessels, has a complete clothing of young epithelial cells around it, ready to spring out and supply new cells for any such break which may occur. These loops are so closely placed that but few new cells are required from each.

The surface of this tissue is arranged for the repair of any such scrapings of its surface which may occur in the chewing of food. This epithelium is continued, and these ends of connective tissue carry the blood supply to the thinnest part of the finest, apparently knife-edge of the crest of the free gingivæ of the labial, buccal and lingual surfaces of the teeth. Then the epithelium is doubled or reflected over the thin margin, and it clothes also the portion of the soft tissue forming the subgingival

space, down to the attachment of the peridental membrane at the gingival line. Within the subgingival space the layer of cells is much thinner, and especially are they much softer and seem to permit, possibly facilitate, the escape of a fluid which continually bathes the subgingival space.

In the last analysis it is seen that the epithelium, taken as a whole, makes up quite a large bulk of the tissue in that part of the gingivæ closely surrounding the teeth on their labial, buccal and lingual surfaces.

Epithelium of the septal gingivæ.

That portion of the surface of the septal gingivæ which is exposed to the friction of food forced over it in the process of mastication is covered with epithelium which in all respects is similar in quality and thickness to that described as belonging to the free gingivæ of the labial, buccal and lingual surfaces. The epithelium is reflected over its margin onto the tissue lining of the subgingival spaces, mesial and distal, in the same manner and with a similar thin coating of a softer quality of epithelium.

In microscopic sections which are cut through the septal gingivæ parallel with the long axis of the tooth mesio-distally, we find a mass of epithelium that has much the appearance of a lobulated gland, buried centrally in the bucco-lingual direction, at a somewhat variable depth. This may be termed the *septal gland.* This lobulated mass varies much in size in different septal gingivæ. Between teeth that lie very closely together at their gingival lines, giving little room for the septal gingivæ, the amount of the mass is so small that it might easily escape observation. When the space between the teeth is wide at the gingival line, this lobulated mass is sometimes a prominent object. Frequently it extends some distance into the septum of the body of the gingivæ.

If this mass of lobulated epithelium is a gland at all, it is a ductless gland of very small dimensions. It is easily seen and its tissue can be well studied; if ducts were there, they could easily be found. I can not now think of this bit of lobulated epithelium as of any special importance. But the regularity of its appearance in this position, and the fact also that, in comparatively recent time, certain ductless glands of small size have been demonstrated to have functions of great importance, should cause us to be careful about casting this matter aside as unimportant. Some one may yet find it to be of physiological importance in the performance of some as yet unknown function.

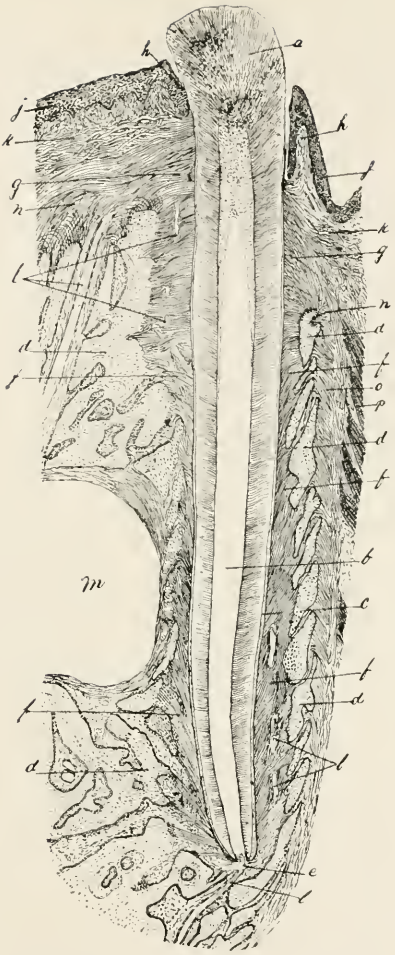

FIG. 25.

FIG. 25. Longitudinal section of a deciduous incisor tooth of kitten with its membrane and alveolus. The actual length of the tooth here reproduced was one-fourth of an inch. a, Crown. b, Pulp chamber and root canal. c, Cementum. d, d, d, d, Alveolar walls. e, Apical space and apical foramen. f, f, f, f, Peridental membrane. g, g, The gingival portion of the peridental membrane. k, Gum tissue. n, n, The periosteum of the outer surface of alveolar wall. h, h, Free gingivæ. j, j, Epithelium. k, k, Coarse fibrous tissue of the gums. l, l, l, Blood vessels traversing the peridental membrane. m, Saculus of permanent tooth. o, Periosteum. p, Attachment of labial muscles. The intention of the illustration is to give a full view of the peridental membrane, and the relations of the tooth, membrane and alveolar wall.

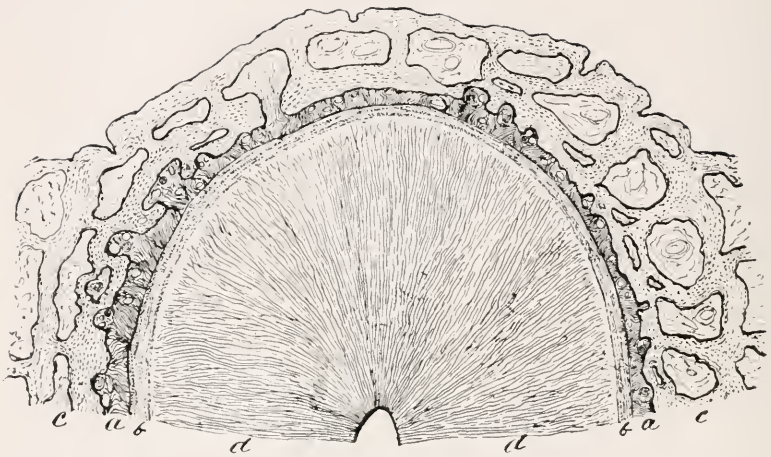

Fig. 26.

Fig. 26. Cross section of cuspid tooth with peridental membrane and alveolar wall cut through the thickened rim at the gingival portion of the alveolar wall, from a man forty years old. The membrane was very thin and firm, and a large piece of the labial wall of the alveolus adhered to the tooth when extracted. It represents an extremely thin peridental membrane, while Figure 98 represents one that may be regarded as thick. a, a, Peridental membrane. b, b, Cementum. c, c, Alveolar process. d, d, Dentin. It will be observed that most of the blood vessels of the peridental membrane lie in depressions in the alveolar wall.

The hormone. In the human body and in the bodies of animals there are a considerable number of ductless glands, large and small, some of which have been carefully studied, and others have not. Of these the spleen is much the largest. Its functions have not been completely made out, but it seems to have a relation to blood formation.

The other ductless glands in their normal state are very much smaller. Perhaps the most important of these, both in the physiological sense and in pathological relations, are the thyroid gland and the suprarenal glands. The former is located about the trachea, and the latter in the suprarenal capsule of the kidney. The thyroid gland is subject to many changes and diseases, the most important of which is exopthalmic goitre, due to the production of a greatly increased amount of secretion, which acts as a systemic poison. The removal of this gland results in a disease called myxedema in adults, or cretinism in children, which may terminate fatally. The central and one lateral lobe are usually removed, also part of the remaining lateral lobe may be removed, with great benefit to patients suffering from exopthalmic goitre. To destroy the suprarenal glands, and they are liable to be destroyed by tuberculosis, is to bring on disease of a wasting character, which ends in death. To remove these bits of tissue by operation, has a like effect.

The brain, the thyroid gland, the suprarenal gland, the liver and muscles form a group of organs whose function is to convert potential into kinetic energy. That is to say, latent energy is converted into motion and heat in response to adequate stimuli, but if these stimuli are too intense, as a result of severe muscular exertion, strong emotion, traumatism, toxins, etc., the cells, especially those of the cerebellum (more specifically, the Purkinje cells) become exhausted and may be permanently disintegrated. Crile's theory of preventing shock consists in weakening or breaking the kinetic chain at any point. There are two ways of breaking this chain. One is by anesthetics which prevent the brain from receiving psychic shock and the other by blocking the nerve tracts to prevent the brain from receiving the shock of traumatism. Both must be employed to carry out Crile's plan.

In the study of these ductless glands, it has been found that each produces a chemic body, or several of them, known as *hormone*, pl. *hormones* (Greek), meaning to excite, to arouse. This chemic substance is different for each ductless gland; that is, each ductless gland secretes or elaborates its own hormone.

These are formed, we may suppose, much as other secretions, but instead of being conveyed to their destination in ducts, they are delivered immediately into the blood stream passing through them, and in this way are carried to the organ with which they are associated, and which they arouse to action. The organ excited in this way may be at a distance from the gland forming the hormone. It seems very curious that just a little bit of ductless lobulated glandular tissue, like the suprarenal glands, should have so important a physiologic function. The drug adrenalin is derived from the suprarenal glands of animals. We do not know but that some one may yet discover that these ductless glands distributed in the septal gingivæ, may also have some important function.

DEVELOPMENT OF THE GINGIVÆ.

Well-known facts warrant the statement that after the intrauterine period, or after the animal of whatever kind has entered upon an independent existence, new tissues needed in growth are not developed until other conditions have rendered their function necessary. Certainly a bony alveolar process, creating an alveolus, is not developed until there is a tooth around which to build it. In the same way, a gingiva does not grow until there is a tooth about which it may entwine its tissue.

When a child is born a *primary alveolar ridge* is found, which is serving as a housing for the developing teeth. When a new tooth makes its way to the surface, this primary alveolar ridge about it is being absorbed to give it exit, and an alveolar process of bone is forming around it. The soft tissue covering of this is simple gum tissue. There is no appearance as yet of the body or processes of the gingivæ. As the crown of the tooth is pushed forward, a considerable part of this gum tissue is absorbed from over it, but a remaining part is pushed aside. Just at the time of the release of the coming tooth from restraint, by the absorption of the bone covering it, the tooth comes forward quickly, so that within a very few days it is finding its occlusion with its fellow of the opposing maxilla. In this last rapid movement any part of gum tissue in the way is simply pushed aside. This often gives the soft tissue about the new tooth a ragged appearance for a few days. These tissues seem to be overfull and swollen; the embrasures may be overfull with the soft tissue standing out of them in festoons. The child may complain of some transient pain from biting food upon this, but

within a few days it is trimmed down to better dimensions by absorption.

The proper forms and structure of the gingivæ have not yet grown. There is not only absorption, which reduces the surface form of the tissue, but also absorptions everywhere *within the tissue* and growth of those tissue forms belonging to the gingivæ proper. This is rapidly built, the new displacing the old, and within a month or two the gingival tissue will have been completed and ready to perform its usual functions. The teeth are apt to present in pairs, of like kind one on either side of the mouth, or four, including the teeth of both upper and lower jaws, and as these erupt the growth of the proper membranes and gingivæ for pair after pair, lower and upper, is proceeding. The changes which take place in these tissues during this period are very rapid. The children, however, if healthy, go on through it all with only a twinge of pain now and then, when they forget and bite food against some bit of ragged tissue freshly pushed aside. It is repetition after repetition of this process, with but little variation, from the time of the eruption of the first of the deciduous teeth until the last of the permanent teeth.

During these growths of the outward forms, the tissues are making interstitial growth. The fibers of the gingival portion of the now scant cementum are growing and forming their groups. These groups include the alveolar crest group, running out to the crest of the alveolar process or to the condensed tissue of the surface of its periosteum, also scattering fibers which extend into the body of the gingivæ. The group of fibers, turning occlusally to form the free gingivæ group, take their places. The fibers of the trans-septal group, which tie the teeth solidly together mesio-distally, grow out through the septal tissues and form their junctions with each other, making these important groups solid and strong. The gum tissue, with its coarse fibrous mat, is changed for a finer network united with the fibers from the peridental membrane. The length of the body of the gingivæ increases as the teeth move farther out from their bony alveoli, and form longer soft tissue alveoli.

Finally, while all of this is in progress, the epithelial covering is being reformed. Indeed this tissue is being actively regenerated during life, but during this time it is rapidly changing its qualities and forms, the cells becoming smaller and more closely interwoven. The mass of cellular elements become more and more thickened. The interdigitation of fine, closely set con-

nective tissue fingers into this epithelium is grown and brought into complete form throughout every part of the tissue.

During this time the outward form is not neglected. As growth proceeds, more and more of the crown of the tooth protrudes through the gingivæ and the depth of the subgingival space is diminished. The changes are grown in the gingival tissue to accomplish this, and go on continuously to the adult period. The tissue is trimmed down here and its fulness increased there until an even smoothness of form is produced in the whole compound of soft and hard tissues which gives a smooth exterior with an intimate network of soft tissues wound about the teeth. This fills perfectly every interstice between and about them in such a way as to prevent lodgments of débris or food occurring at any point.

Such is the picture of a perfect development. Unfortunately, we do not always find it so perfect, nor do we always employ the best means to correct and smooth over and improve the imperfect points, or protect those which are good, from abuse.

The fibers from the peridental membrane distributed in these tissues serve to bind the whole group into a solid mass, or into a mass that has a very powerful controlling effect upon the establishment of the dental arch along right lines, and maintaining it in this form. The development of any inflammation in this tissue serves to soften the fibers and causes them to stretch more easily, or even causes them to swell and occupy too much space. This often throws this tissue out of form, interfering with its close adaptation to the teeth, and roughening the margins of the free gingivæ, causing them to receive lodgments instead of shedding such material away during the process of mastication, as they should do. This will be studied more in detail later on.

The subgingival spaces. The subgingival spaces have been sufficiently defined. Any particular subgingival space will be located by naming the tooth to which it belongs, as, the lower left central incisor subgingival space. The parts may be designated by naming the surfaces of the tooth which are covered, as the mesial, distal, lingual, labial or buccal subgingival spaces, for each tooth. If it should become necessary, and it will, we can particularize almost any part of the subgingival space, as the subgingival space at the disto-buccal angle of the upper first bicuspid, etc. While we name these various parts, it should be particularly noted that the subgingival space really encircles the

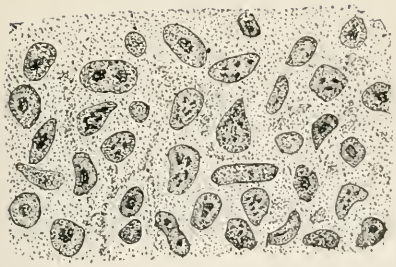

FIG. 27.

FIG. 28.

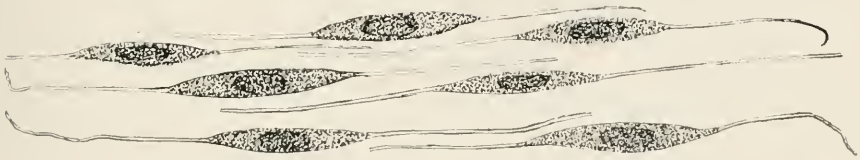

FIG. 29.

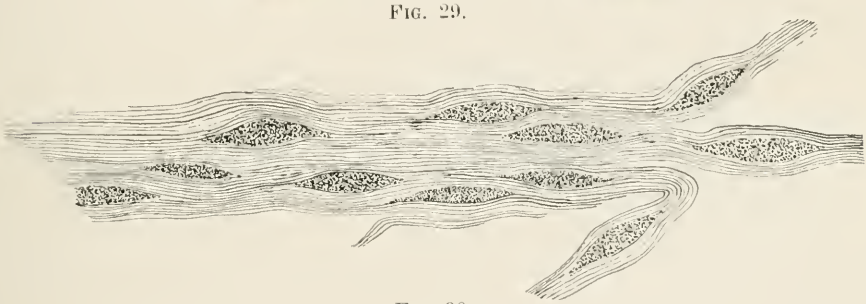

FIG. 30.

FIG. 31.

FIGS. 27 TO 42. A series illustrating the growth of connective tissue.

FIG. 27. Embryonal connective tissue in an early stage of development, showing the cellular elements imbedded in the ground substance.

FIG. 28. The same, a little more developed, showing the cellular elements lengthening in a common direction.

FIG. 29. The cells, developed in spindle forms, fibroblasts with long filaments extending from either end.

FIG. 30. The developed white fibrous tissue.

FIG. 31. Older white fibrous tissue, in which the cells are no longer seen, and showing the wave-like course of the fibers.

FIG. 32.

FIG. 33.

FIG. 34.

FIG. 35.

FIGS. 27 TO 42. A series illustrating the growth of connective tissue.

FIG. 32. Coarse white fibers, made up of bundles of the fine fibers, and showing the mode of division by the splitting off of a portion of the fibers of the bundle.

FIG. 33. Coarse fiber breaking up into fine fibers.

FIG. 34. Cross sections of coarse fibers showing some of their various forms.

FIG. 35. Tissue of the dental pulp, in which the development of the cells is not followed by any considerable formation of fibers.

FIG. 36.

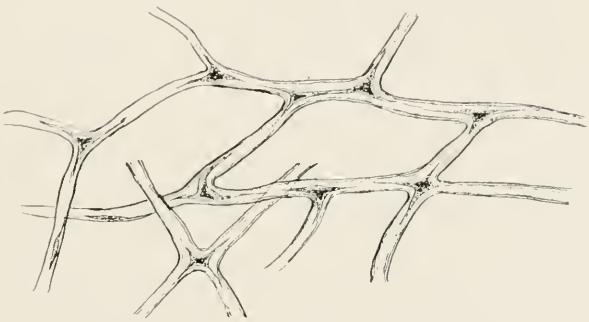

FIG. 37.

FIG. 38.

FIGS. 27 TO 42. A series illustrating the growth of connective tissue.

FIG. 36. Connective tissue cells from which reticular fibers are developed.

FIG. 37. Reticular fibers, showing the mode of division and the multipolar, or irregular star forms of the cells at the divisions.

FIG. 38. Cross sections of the reticular fibers, showing some of their forms.

FIG. 39.

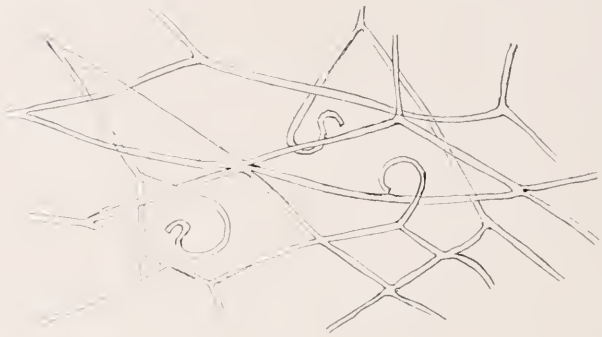

FIG. 40.

FIG. 41.

FIG. 42.

FIGS. 27 TO 42. A series illustrating the growth of connective tissue.

FIG. 39. Network of elastic fibers teased out from elastic tendon, and showing the usual mode of division.

FIG. 40. Network of elastic fibers from the point of reflection of the mucous membrane of the lip from the gums.

FIG. 41. Elastic fibers, showing their disposition to curl up when cut or broken.

FIG. 42. Cross sections of elastic fibers, showing their forms as seen in a group passing between coarse white fibers.

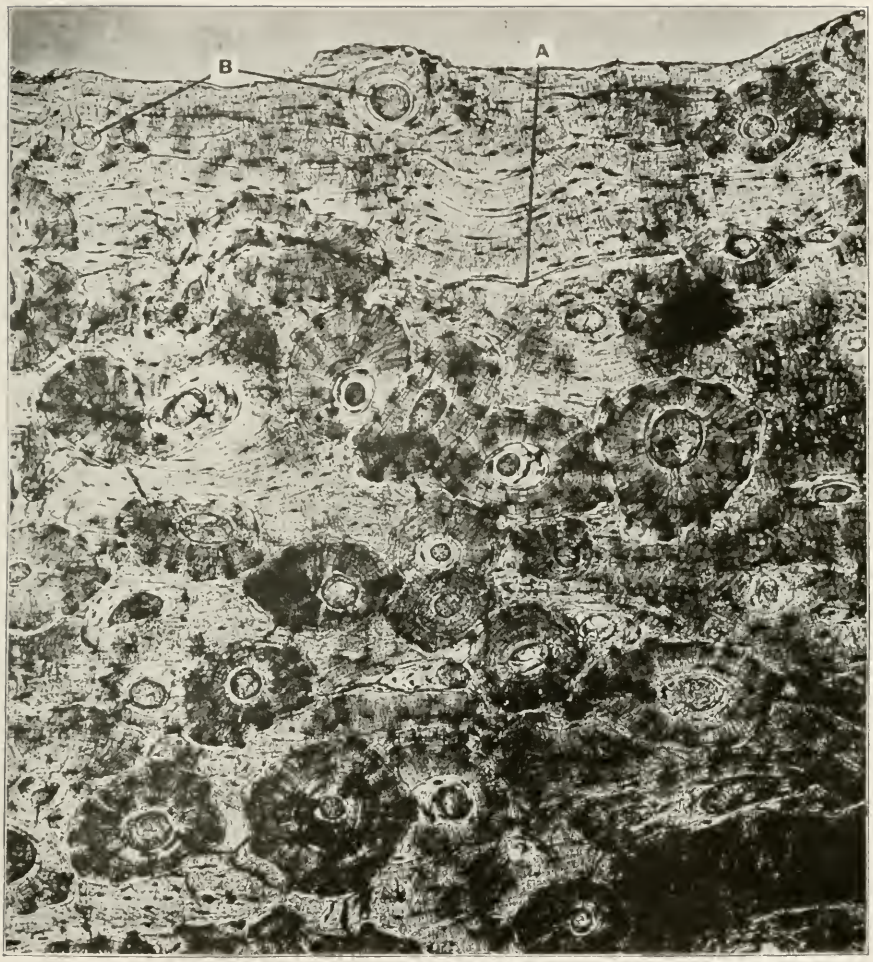

FIG. 43.

FIG. 43. A photomicrograph from a cross section of bone from the human femur from a young person. A, This line crosses laminæ of subperiosteal bone. B, These lines point out Haversian system bone. These Haversian systems, that are seen to form the bulk of tissue, are formed by the absorption of the original subperiosteal bone and building in the Haversian system bone.

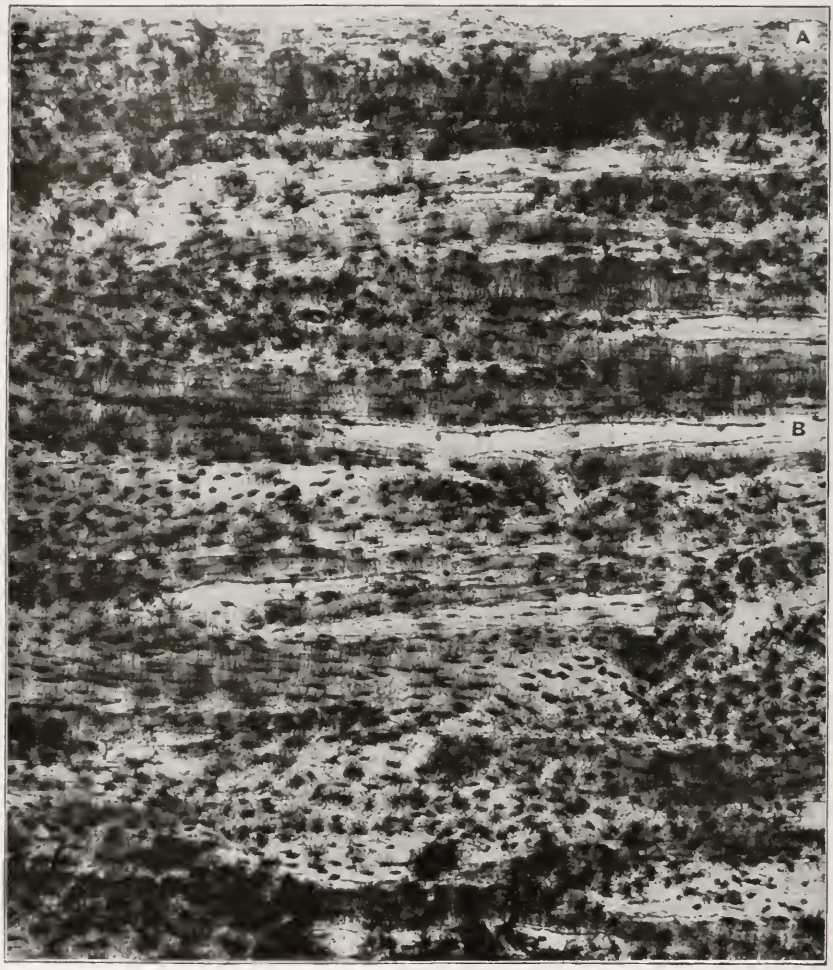

FIG. 44.

FIG. 44. Lengthwise section from the same bone, as illustrated in Figure 43, showing the Haversian systems and their canals cut lengthwise. A, Subperiosteal bone. B, A Haversian canal.

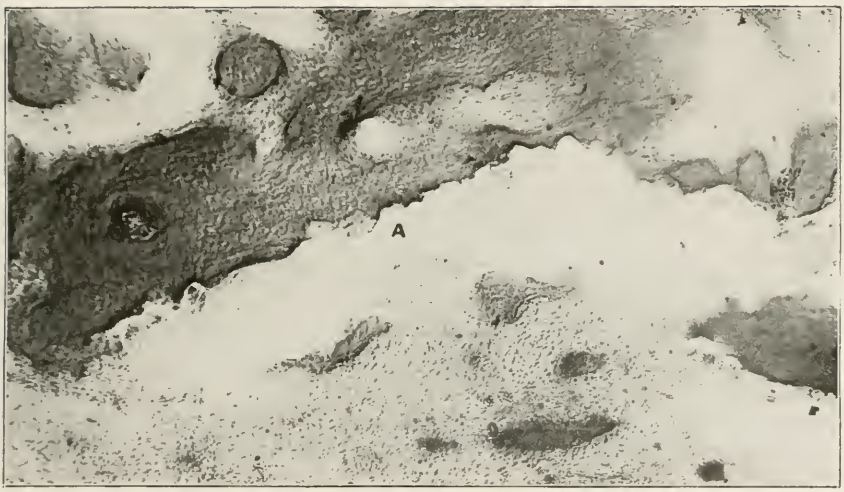

Fig. 45.

Fig. 45. A photomicrograph of bone in process of absorption. A, Line of absorption showing the lacunæ of Howship.

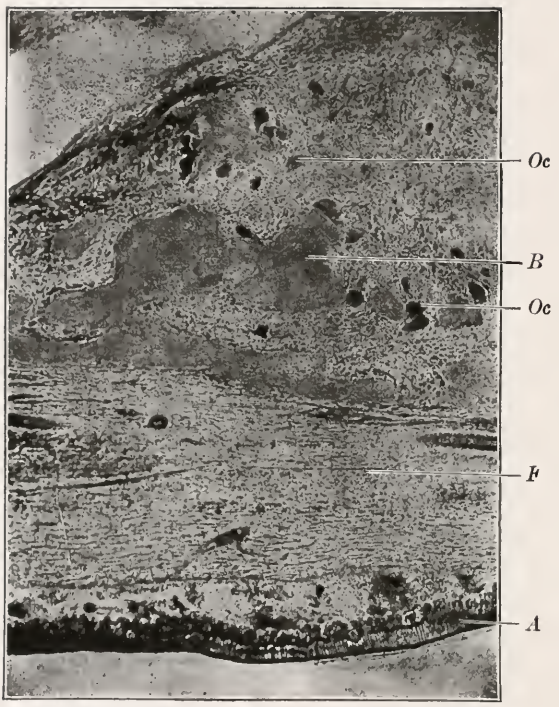

FIG. 46.

FIG. 46. Osteoclast absorption of bone over permanent tooth: oc, Osteoclasts. B, Bone of crypt wall. F, Fibrous tissue of follicle wall. A, Ameloblasts. *Noyes.*

entire circumference of the tooth without break. We divide it into parts by these names, for convenience in description. The labial or buccal subgingival space means simply that part of the general subgingival space completely surrounding the tooth, which covers the labial or buccal surface.

It will be noted from the above that I have made use of the same terms in naming the parts of the gingivæ and subgingival space as have been previously used in the descriptions of the surfaces of the teeth.

Exploration of the subgingival spaces. Some of the worst forms of disease of the peridental membranes begin in the depths of the subgingival spaces about the attachment of the tissue to the teeth. Therefore, the exploration of these spaces is of first importance as a preparation for the early detection of diseases of this character. This exploration may be made with any ordinary thin, flat scaling instrument, the sharp angles and working edge of which have been rounded off. Instruments made especially for this purpose are to be preferred. These will be described, and detailed instructions for their use given, under the consideration of examinations of the mouth.

In the examination of a number of persons, ranging from eight to forty years, one will gain a correct idea of the changes which occur in the depth of the gingivæ as age advances, which will be very useful, and can not be so well learned in any other way. Experience in subgingival examinations will enable one to detect the beginnings of disease at the attachment of the peridental membrane. For this purpose, such a course of experimental study is actually essential. One should examine hundreds of cases of normal gingivæ before he is ready to study diseased conditions of this tissue.

In making such a series of examinations, one will obtain much other valuable information regarding the subgingival spaces. It will be found that the distance from the incisal edge of the central incisor to the gingival line is much greater upon the labial surface than on the proximal surface, yet the gingivæ will be longest on the proximal surface. This is because of the form or direction of the gingival line around the incisor tooth. This line is curved upon the labial surface with the concavity toward the incisal edge of the tooth. It passes around the proximal surfaces in a curved line which presents its convexity toward the incisal edge of the tooth. This form of the line of attachment, the gingival line, is common to the incisor teeth and the mesial surfaces of the cuspids, above and below. The distal

surface of the cuspid usually has only a slight curve toward the incisal. On the bicuspids and molars the usual course of the gingival line is more nearly directly around the tooth. (See Figures 15 to 24.)

On some teeth the gingival line has irregularities which one should be able to recognize. These irregularities consist in what I have called bridges and pitfalls. As the instrument is passed around the tooth feeling the attachment, it may strike a bridge, a point where the attachment is higher on the crown. At this point there is a spot of thickened cementum that has lapped a little more than usual upon the enamel. On carefully lifting the end of the instrument onto this, it is often found to be of only slight extent and then again drops to the general level. On the other hand, the instrument may drop into a depression in the line around the tooth. These also may be very narrow, after which the line resumes the general level. It is essential that one should become well acquainted with these, so that he may not mistake them for beginning pus pockets.

FUNCTIONS OF THE GINGIVÆ.

A PROTECTIVE TISSUE. The function of the gingivæ of first importance is that of a protective tissue. This is a passive function exerted through the form and solidity of its structure, as it is fitted and wound about every part of the teeth, filling smoothly all interstices and shielding the tissues beneath. When this form is good the membranes of the teeth will be well protected from injury. When this form is not good, these will be more liable to injury.

The hard tissues of the teeth become useless without their soft tissue investment. Their usefulness depends directly upon the strength and healthfulness of that investment. It is not enough that the gingivæ by the aid of the bony alveolar process hold the teeth strongly in their positions. The forms which they entwine about the teeth must be such as will shed off the débris of mastication and prevent all lodgments about them, which in their decomposition would give rise to offensive and disease-producing compounds. To do this, every part of the surface of the gingivæ must be of such form as to fill all interstices full enough, but not too full, and thus be effective for both cleanliness and accommodation of food movements in the acts of mastication and deglutition.

The outward form of the gingivæ in and of itself, is of the utmost importance. The maintenance of this is one of the first

elements of good service in dentistry. Any deviation from the best form constitutes a barrier to the health of the teeth and their investment — the gingivæ and the peridental membranes. In the past, dentists have treated the gingivæ as unimportant, and have not studied them. Often they have wantonly destroyed them, especially the septal gingivæ, in connection with the filling of proximal cavities. Now we are finding the reward in an increase of disease beginning at the gingival line.

For a number of years I have, whenever opportunity offered, studied conditions controlling deposits upon artificial dentures. I have worn a plate myself, and often have had several, upon which I could study places of deposit at will. In every case a depression has meant a place of deposit of some kind. In some of these food lodges, remains and is decomposed; in others perhaps calculus gathers; in others there may be a cheesy deposit, or some form of débris. Deposits upon artificial dentures are in different positions from deposits in the normal mouth. The whole surface of the plate may become susceptible of receiving and holding deposits.

In the mouth that is normal there is but one deposit on the soft tissues, and that is mucus which renders the surface of the epithelium slippery, so that material of almost any kind glides easily. This is normal and is present in every mouth. It is, however, sometimes in abnormally large quantity, and sometimes in scant quantity. This seems to have little in common with other deposits. A short gingiva which causes a depression about a tooth generally makes a place for the lodgment of calculus or cheesiform deposits. True, it makes some difference where it is located. If on the buccal surfaces of the molars, it is certain to catch calculus if any at all comes into the mouth. If there is much calculus coming into the mouth, it will catch some of it, no matter where it is located. The same is true to even a greater extent with the cheesiform deposits, which will be studied later.

One should understand distinctly that these deposits, other than mucus, may occur anywhere, where there are hard tissues or mechanical appliances on which they can lodge. In the mouth the deposit can not occur except on the teeth or some hard substance placed in the mouth. It never adheres to the mucous membranes or other soft tissues. If present, as sometimes occurs, in soft tissue cavities, as in the tonsils or nose, the initial deposit is on some hard or dead substance which furnishes a nidus. A beginning is never made on living soft tissue. This

may be regarded as a statement in pathology, but its basis belongs to physiology. The mucus is practically the only substance that is deposited upon the soft tissues. This deposit renders these tissues, and all other tissues of the mouth indeed, slippery, and in this way performs a very important function.

Maintenance of the teeth in the line of the arch. A second function of the gingivæ, perhaps in a degree a part of the first, is the maintenance of the teeth in the line of the arch. The influence of the bone forming the alveolar process has been much overrated in its importance in maintaining the teeth in their positions. Hard and rigid as the bones of the skeleton seem in the dried state, bone is a very plastic tissue during life, and is bent about in almost any direction by a constant artificial pull. In the treatment of clubfeet I have seen the bones of the lower leg bent much out of their normal shape by a comparatively light continuous pull upon them. This was effected by light rubber straps attached to the upper part of the leg by adhesive plaster, and reaching to the feet, to make tension in certain directions. When these straps are released and the muscles and nonmuscular tissues of the connective tissue group resume their functions, these bones quickly return to their normal form. The bones are good as holding against a stress suddenly applied and then released, but not for a continuous stress out of the normal directions. This is especially true when there is an interference with the normal action of the soft tissues for the time.

When a lower first molar is extracted at a certain time of life, its alveolus is filled with bone, and the alveolar process, as such, disappears. The gingivæ are swept away, and a cicatricial tissue is formed in the space to which the ends of the fibers of the trans-septal group are fastened. The shrinkage of this cicatrix and the pull of the trans-septal group of fibers drags the second and third lower molars to the mesial and tips them mesially until their occlusal surfaces do not meet their fellows correctly.

These teeth are literally dragged through the bone, endwise of the bone, where there is no possible chance for the bone to be bent away. The solid bone must be moved, or when it can not be moved in substance, it will be moved by absorption in one direction, and building in, in the other. It will not stand against a connective tissue constancy of stress. We have been long in finding that the connective tissue group, other than active muscles, has a great function in directing the building of the

body, holding organs in their places in health, and bringing them back to place when the correction of conditions will allow them freedom of action. These, indeed, are the most active of the tissues in maintaining the phylogenetic play of forces in shaping, trimming, forming and maintaining the development of the body in its general ancestral forms, and yet with the finest sense of ontogenetic development, or the shaping of the individual in all of its parts. There is no place in the human body where we find as fine examples of this play at control of form by the non-muscular connective tissue as in the gingivæ, or so much harm from its influence when the conditions have given them a wrong direction. This will necessarily come into discussion often in pathological studies of the influence of the various tissues.

THE CEMENTUM, PERIDENTAL MEMBRANE AND ALVEOLAR PROCESS

ILLUSTRATIONS: FIGURES 25-121.

The cementum and peridental membrane and the correlation of these two tissues in health and disease are of the highest degree of importance, yet of all of the dental tissues, these are the least well understood.

It is quite essential that one should have a clear understanding of the physiology of the several tissues of the teeth, and their physiological and pathological relations to the tissues with which they are directly connected. One should know how these act and react toward each other, the limitations of their powers in recuperation following disease or accident, and the more general questions along this line. If these are well understood, it will be comparatively easy to comprehend the pathological conditions, their symptomatology, and what may and what may not be accomplished in treatment. Knowledge of what can not be done is as important in practice as knowledge of what can be done. Many dentists are losing time and prestige in trying over and over again to do things which the history of cases has demonstrated to be impossible. We should know the history of these efforts and failures, and their meaning in pathology. Slowly, possibly very slowly, we will find ways to do things which we can not do now. We should ever be on the watch for improvement, but should be very careful about pinning faith to fancies in the treatment of disease.

Histological studies of the peridental membrane. In the years preceding the publication of the American System of Dentistry (1886), there was considerable speculation as to the structure of the peridental membrane. There were, however, no studies of this tissue available which seemed to me to be at all sufficient, or which bore the stamp of real histological work. When I was called upon to write the article on the cementum and peridental membrane, and their diseases, for that publication, I undertook the histological study of these tissues along with other work with which I was unusually busy. When my copy was otherwise ready, I found my studies of the histology

so hopelessly behind that I wrote a very short description of some of the principal features, and forwarded my copy to the printer. But others were so far behind in their work that the final completion of the book was much delayed. In the meantime I had found the facts on which I could have written the histology complete. I was glad to find later that my short and insufficient description of the membrane contained no serious errors.

My later studies of the histology of the peridental membrane and the comparative study of the periosteum in different parts of the body were embodied in a series of articles published in the Dental Review, beginning with its first issue in November, 1886, and continuing in 1887 until completed. As soon as this publication was completed, the copy was revised and published in book form under the title of "The Periosteum and Peridental Membrane," 1887. Figures 27 to 42, illustrating the growth of connective tissue, and Figures 47 to 65, illustrating the growth of bone, are reproduced from this book. Very slowly the facts developed in these studies are finding their way into our better text-books, as yet insufficiently stated, but with improvement as the years go by. The very concise statements of the histological structure, with excellent illustrations, by Dr. F. B. Noyes, in his book on dental histology, published in 1912, are assisting materially in spreading correct information.

During the past few years, there have been a number of articles by German scientific observers relative to the cellular elements in the peridental membrane. Many of these have been written by able histologists, and add much of accurate knowledge of these tissues, both in their normal condition and in their pathological changes. Reference will be made to these studies in the consideration of the specialized cells of the peridental membrane and also in cyst formation.

The Cementum.

ILLUSTRATIONS: FIGURES 43-92.

The cementum covers the root portion of the tooth, enclosing the dentin, and usually slightly overlaps the gingival portion of the enamel. The attachment of the peridental membrane is therefore to the cementum.

The cementum is a specialized tissue. Nothing like it exists elsewhere in the animal body. It is in every respect a passive tissue. It does not originate any form of physiological activity.

It does not build itself, nor repair injuries to its own tissue. It is laid down on the dentin by the peridental membrane very much as subperiosteal bone is built by the periosteum. It is much like bone, and has in its substance corpuscles very like the bone corpuscles. Especially, it closely resembles subperiosteal bone in its histological content. But the corpuscles are usually fewer and less regularly placed. In some specimens, however, the cement corpuscles are plentiful. In this, different specimens vary widely. The cementum is much thicker toward the apex of the root, and thins away toward the gingival line, which it forms by lapping slightly on the margin of the enamel.

Differences between cementum and bone.

The point in which cementum differs most widely from bone is in the absence of a blood vascular system. In bone every part of the tissue is within the sphere of the circulation of red blood, and, without aid from adjacent tissues, is subject to absorption and perfect rebuilding of its own tissues at any time. It has this power within its own tissue. Also subperiosteal bone is cut away by absorption and rebuilt as Haversian bone, which has numerous channels conveying arteries, veins and nerves.* (See Figures 43, 44, 45 and 46.) Cementum has none of these whatever. It has no circulation of red blood in any form. It is therefore dependent upon the peridental membrane for the maintenance of the life of its cement corpuscles.

Cementum does not repair injuries.

Cementum has not in itself any power of repairing injuries to its tissue. When stripped of its peridental membrane it becomes a dead tissue, no matter if the pulp of the tooth is alive. The tissue of the cementum has no power of initiating or carrying forward any reparatory process whatever in the absence of the soft tissues around it, or when these have been parted from it by suppuration.

Cementum subject to absorption.

The otherwise normal cementum is very subject to absorptions. These begin upon the outside, next to the peridental membrane, and extend inward or laterally from that beginning. This absorption is the true physiological process of the removal of the roots of the deciduous teeth in the shedding of these in

* "The Growth of Bone," by William Macewen, F.R.S., published in Glasgow in 1912, is a splendid work which gives a much broader view of the growth of bone and its powers than it is possible for me to give here.

FIG. 47.

FIG. 48.

FIGS. 47 TO 65. A series illustrating the growth of bone.

FIG. 47. Non-attached periosteum from the shaft of the femur of the kitten. B, Bone. O, Layer of osteoblasts. In the central portion of the figure they have been pulled slightly away from the bone, displaying the processes to advantage. It will be observed that the fibers of the periosteum do not enter the bone. a, Inner layer of fine white fibrous tissue (osteogenetic layer) showing the nuclei of the fibroblasts and a number of developing connective tissue cells, which probably become osteoblasts. c, Outer layer, or coarse fibrous layer, in which fusiform fibroblasts are also rendered apparent by double staining with hematoxylin and carmine. d, Some remains of the reticular tissue connecting the superimposed tissue with the periosteum.

FIG. 48. Attached periosteum from beneath the attachment of the muscles of the lower lip of the sheep. a, Bone. B, Osteoblasts, with the fibers emerging from the bone between them. c, Inner layer with fibers decussating and joining the inner side of the coarse fibrous layer in opposite directions. This is rather an unusual form of this layer of the periosteum. D, Coarse, fibrous layer. E, Attachment of muscular fibers.

FIG. 49.

FIG. 50.

FIGS. 47 TO 65. A series illustrating the growth of bone.

FIG. 49. Periosteum from the shaft of the tibia of the pig, lengthwise section, showing the complex arrangement of fibers in the coarse or outer fibrous layer which sometimes occurs under muscles that perform sliding movements. B, Bone. O, Layer of osteoblasts. The tissue has been pulled slightly away from the bone in mounting the section, and part of the osteoblasts have clung to the bone, some have clung to the tissues, while others are suspended midway, their processes clinging to each. a, Layer of fine fibers. Inner or osteogenetic layer of the periosteum. b, First lamella of the coarse or outer fibrous layer, the fibers of which are, in this case, circumferential, exposing the cut ends. It will be observed that there are ten lamellæ in the make-up of the outer layer, the lengthwise and circumferential fibers alternating. Those marked f, and i, are very delicate ribbon-like forms, which have shifted from their normal position in the mounting of the section, so as to present their sides to view instead of their ends, thus displaying their structure to advantage. The illustration shows how readily separable these lamellæ are. l, Reticular tissue.

FIG. 50. Periosteum from the lower end of the femur of the kitten at a point where the enlarged end next the joint is being trimmed down for the elongation of the shaft, showing the fibers of the periosteum included in, or entering the bone, forming its attachment, also the absence of osteoblasts and the presence of osteoclasts by which the outer portions of the bone are being removed. B, Bone. c, Osteogenetic, or inner layer of periosteum. d, Outer layer, a part of which seems to have been torn away. E, A few circumferential fibers. f, f, f, Osteoblasts lying in the lacunæ of Howship, or excavations in the bone made by these cells.

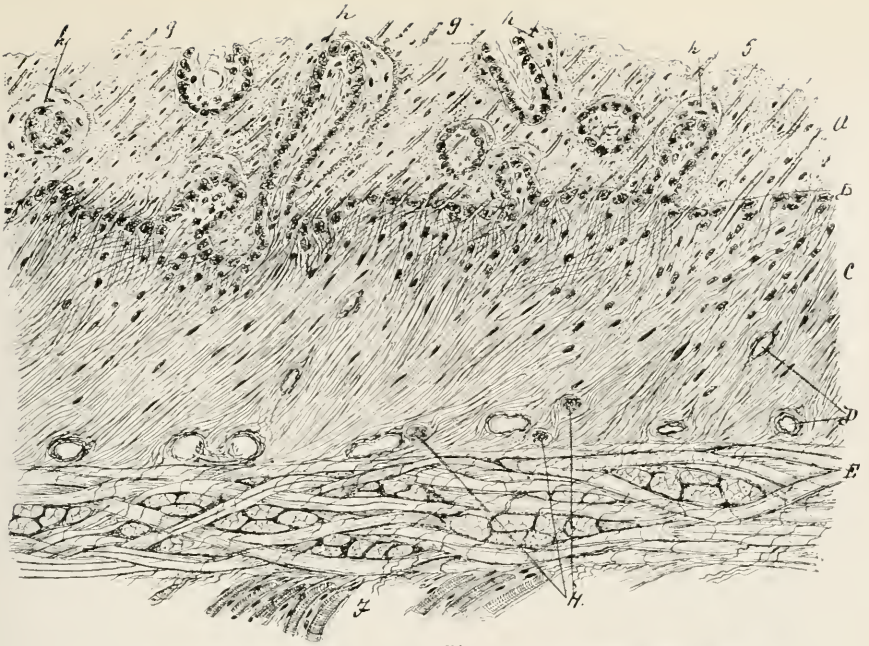

FIG. 51.

FIG. 52.

FIGS. 47 TO 65. A series illustrating the growth of bone.

FIG. 51. The more usual form of the attached periosteum. A, Bone, showing the residual fibers (penetrating fibers of Sharpey) within its substance and passing out between the osteoblasts B, and breaking up into fine fibers, which form the internal layer of the periosteum. These are also seen protruding from the broken margins of the section at g, g, g. D, Blood vessels which are cut across. They occur mostly in the inner layer, very close to the under side of the outer layer. H, Small nerve bundles. F, Attachment of muscular fibers. It will be noted that the Haversian canals at h, h, h, h, and at other points, are filling up with bone which has no residual fibers.

FIG. 52. A photomicrograph of an attached periosteum similar to Fig. 51. From the alveolar process of a sheep. *Noyes.*

FIG. 53.

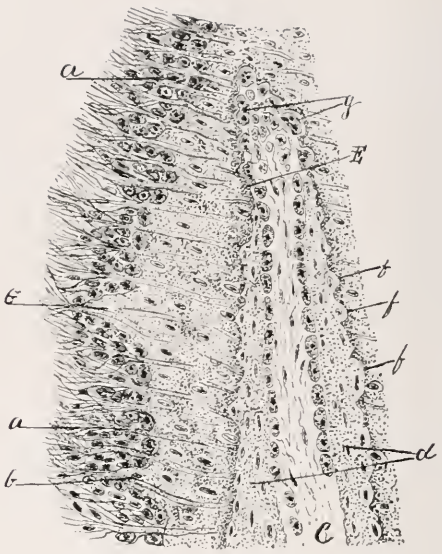

FIG. 54.

FIGS. 47 TO 65. A series illustrating the growth of bone.

FIG. 53. Bone, with portion of inner layer of attached periosteum, and penetrating fibers. The section is cut across the Haversian canals, and it shows the manner of the formation of these in the surface of the growing bone at a, a, by the upward growth of spiculæ of bone which then spread out and join with others, thus bridging over and forming canals. At b, b, b, b, four Haversian canals are seen lined with osteoblasts. Around each of these, fresh bone is being deposited, which may be recognized by a slight difference in shade, but especially by the fact that the bone corpuscles lie in a different position from others in their neighborhood, and the fact that this bone has no residual fibers. It should be noted that this formation of canals immensely increases the area upon which osteoblasts may build.

FIG. 54. Bone, with a more solid growth of surface, and with osteoblasts much crowded between the fibers of the periosteum as they emerge from the bone. Only a part of the inner layer of periosteum is shown. a, a, Osteoblasts several layers deep between the fibers of the periosteum. b, b, Spiculæ of bone growing up into the periosteum, apparently following the line of a particular fiber. C, Haversian canal that seems to have been excavated in the bone, and is being filled by deposit of new bone on its walls. This new deposit of bone is distinguished by a somewhat lighter shade, and the difference in the direction of the long axis of the bone corpuscles, and the absence of residual fibers. Osteoblasts appear in this portion of the canal. The margins of the secondary formation show the bay-like forms usual in the absorption of bone. Above the line drawn at E, no secondary bone is found, and osteoclasts, g, g, are seen instead of osteoblasts. In this portion the excavation is going on. In this way the bone, with residual fibers, is removed and bone deposited in which these do not appear.

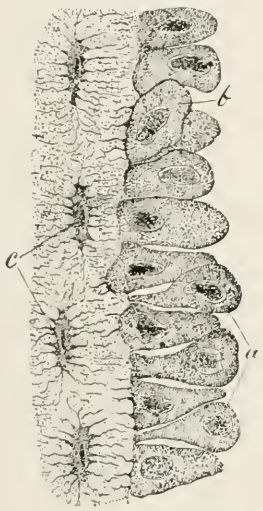

FIG. 55.

FIG. 56.

FIGS. 47 TO 65. A series illustrating the growth of bone.

FIG. 55. Margin of growing bone upon which the osteoblasts are very much crowded. a, Osteoblasts reaching to the surface of the bone by extending process-like prolongations. b, A cell that seems to be flattening down upon the surface of the bone. c, Bone corpuscles, the processes of which are seen radiating in the bone matrix. Processes are also seen extending into the bone from some of the osteoblasts.

FIG. 56. Cross section of a young growing bone, showing the Haversian canals and the plan of their subperiosteal formation. a, Outer layer of periosteum. b, Inner layer of periosteum. c, c, Spiculæ of bone growing outward into the tissue of the inner layer of periosteum. d, Other and older spiculæ spreading out at their summits, forming portions of arches. e, Other spiculæ, the arches of which are about closing to form Haversian canals. f, Complete Haversian canals, many of which are seen in the illustration.

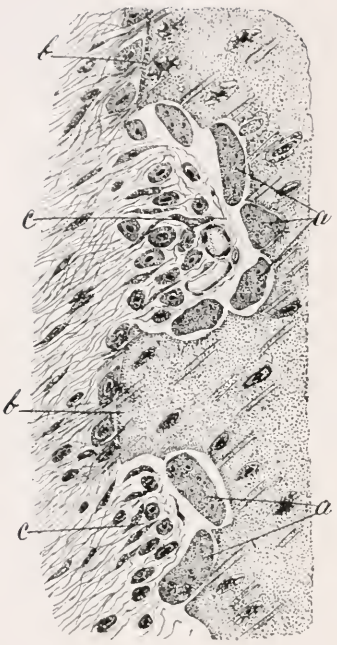

Fig. 57.

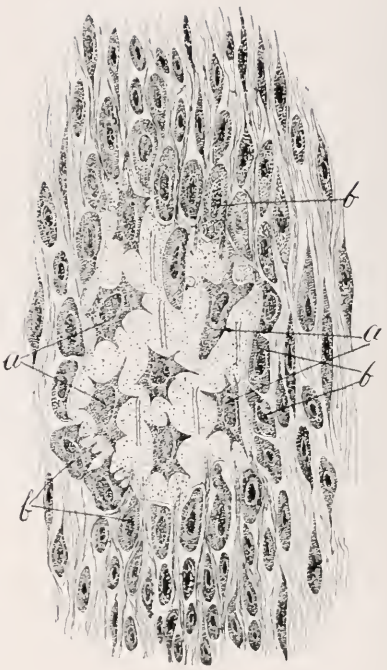

Fig. 58.

Figs. 47 to 65. A series illustrating the growth of bone.

Fig. 57. Absorption of bone under attached periosteum. a, a, Osteoclasts lying in deep excavations in the surface of the bone. b, b, Surface of bone, showing the fibers of the periosteum implanted in it. Residual fibers appear in the bone. It will be noted that these fibers are removed with the bone by the absorptive process. c, c, Masses of embryonic tissue filling the areas formed by the absorption.

Fig. 58. Intra-membranous formation of bone. An island of bony deposit. a, a, Bone corpuscles. b, b, Osteoblasts. It will be seen that these lie between the fibers of the membrane, so that in certain positions the osteoblasts lie with their ends to the forming bone. For the most part the long axes of the bone corpuscles have a similar direction.

FIG. 59.

FIG. 60.

FIGS. 47 TO 65. A series illustrating the growth of bone.

FIG. 59. Growth of bone under the attachment of the Tendo Achillis in a young lamb. A, Fibers of tendon partially converted into fibro-cartilage. The cartilage cells are seen mostly between the tendon fibers. B, B, and c, c, c, Canals advancing from the bone beneath into the tendon. D, D, D, Bone deposited upon the walls of the canals forming Haversian systems laid upon, or among the tendon fibers. E, Portions of the tendon fibers still remaining deep among the Haversian systems of bone.

FIG. 60. A, Single canal as shown at b, Fig. 59, very much enlarged. a, a, Cartilage. b, b, Tissue of canal. c, Blood vessel. d, d, Bone. e, e, Osteoblasts. f, f, Chondroclasts. In both these figures the bay-like excavations of the absorption cells are seen in the canals, and at the margins of the bone deposited in these.

Fig. 61.

Figs. 47 to 65. A series illustrating the growth of bone.

Fig. 61. The changes which occur in diaphysial intra-cartilaginous formation of bone. a, Cartilage unchanged. At b, the cells have become smaller and have fallen into rows. At c, the cells are enlarged in their short diameters, or in the direction of the length of the shaft of the bone. At d, the growth of the cells has reached its limit. The matrix begins to calcify. At e, the capsules of the cells are opened by the advance of the absorbent tissue. f, Area of the formation of bone. g, Apparently some glutinous remains of the cell body clinging to the walls of the capsule. h, Small round marrow cells. p, p, p, Remains of the cartilage matrix. j, Osteoblasts applied to the remains of cartilage matrix, but no bone is seen. k, k, k, Osteoblasts and a layer of bone deposited on the remains of cartilage matrix. m, m, m, m, Blood vessels. n, Capsule which seems to have been just opened and the marrow cells seen in the act of crowding into it. o, Fusiform cells. Many of these appear in this portion of the figure, and seem peculiar to this location.

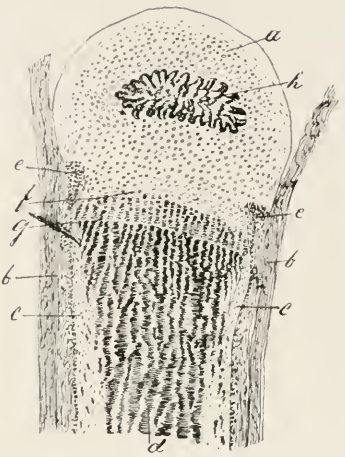

Fig. 62.

Fig. 63.

Figs. 47 to 65. A series illustrating the growth of bone.

Fig. 62. Central section of the head, and portion of the shaft, of the tibia from young kitten, showing diaphysial intra-cartilaginous formation of the bone at d, and the beginning of the epiphysial at h. a, Cartilaginous head of bone. b, b, Periosteum. c, c, Layer of subperiosteal bone. e, Periosteal notch; the point to which the subperiosteal formation of bone extends. f, Beginning of change in the cartilage cells where they form rows. g, Line of absorption of the cartilage. At d, the darkened portion reaching up to the line g, shows the portion occupied by the bone marrow, and the light portions the bone formed.

Fig. 63. Supplement to Fig. 61, taken from another portion of the section and showing the marrow cells applied closely to the walls of the capsules next to be opened. a, Cartilage. b, Fusiform cells filling closely the last capsule opened in that row. c, c, Round, marrow cells filling other capsules in the same manner. d, Unabsorbed remains of cartilage matrix.

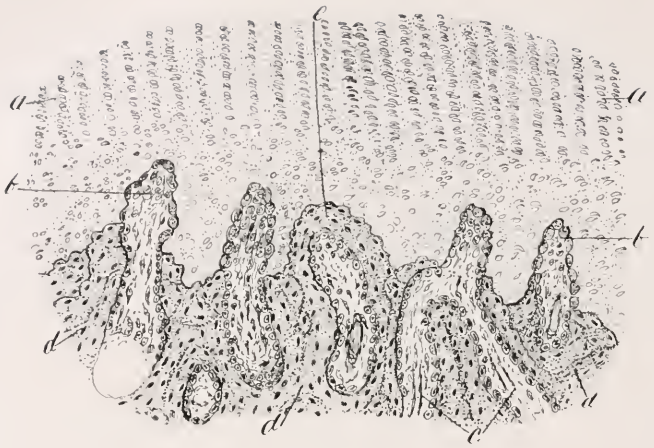

FIG. 64.

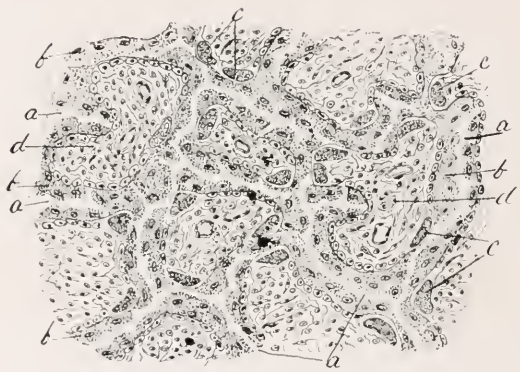

FIG. 65.

FIGS. 47 TO 65. A series illustrating the growth of bone.

FIG. 64. Epiphysial intra-cartilaginous formation of bone from head of tibia of young lamb. a, a, Cartilage, the cells of which have fallen into rows, but have become scattered between the letters a, and b, b. b, b, Haversian canals advanced from the bone into the cartilage. It should be noticed that these are lined with chondroclasts where the absorption of cartilage is in progress, and with osteoblasts when bone is being deposited. c, Blood vessels. d, d, d, Bone, which is extended into the cartilage by the filling of the canals formed by absorption as shown at c.

FIG. 65. From a cross section of a rib of a young kitten at a little distance (boneward) from the change from cartilage to bone showing the large Haversian canals with the remains of the cartilage matrix enveloped in the bone formed. a, a, a, a. Remains of cartilage matrix, which, in the figure, is left white. b, b, b, b, Bone deposited on remains of cartilage matrix, and generally covered with osteoblasts, but at c, c, c, c, and other points, osteoclasts are quite plentifully distributed. While in one part bone is being deposited, in another it is being removed, and in the end all the cartilage matrix disappears.

Fig. 66.

Fig. 66. A photomicrograph of a portion of the root and peridental membrane of a tooth in which an absorption has been repaired by a new growth of cementum. D, Dentin. C, Cementum. P, Peridental membrane. R, New cementum built in, in repair of an injury by absorption.

FIG. 67.

FIG. 67. Photomicrograph of a cross section of the root of a tooth in which absorption is in progress. A, Line of absorption, showing the usual notched appearance known as the lacunæ of Howship. D, Dentin. C, Cementum. P, Peridental membrane. The pulp chamber appears in the left-hand part. The dento-cemental junction appears between the letters C and D.

order that the permanent teeth may take their places. During the absorption of the roots of the deciduous teeth, it often occurs that a beginning absorption is repaired by the deposit of new cementum by the cementoblasts of the peridental membrane, filling up the breach made, either partially or completely. These repairs often occur after the absorption has penetrated the dentin to considerable depths. No matter what the depth of the absorption in dentin, the repair of the whole depth is always made by a deposit of cementum, never by building in dentin. (See Figures 66 to 74.) There is nothing like an inflammatory movement connected with these absorptions. They are always effected by the formation of an aggregation of specialized cells, the cementoblasts. They do not differ materially from absorptions elsewhere in the body, as in the bones. In the bones, absorptions for the formation of new Haversian canals may always be found in progress in young subjects. The characters of the cellular groups effecting the absorption seem to be much the same, whether the results of the absorption be regarded as physiological or pathological.

Absorption of roots of permanent teeth.

Absorptions of portions of the cementum, often penetrating into the dentin also, occasionally occur in the roots of the permanent teeth. In part, this may be regarded as normal. In the movements of the teeth which occur, for instance, because of the loss of one of their number, the fibers of the peridental membrane are sometimes cut away over a space, and a part or all of the cementum removed, and possibly some part of the dentin also. When the movement of the tooth has been accommodated, the space will again be covered with new cementum, into which the principal fibers of the peridental membrane will again be attached. This can not be regarded as pathological. Much more generally these movements are accommodated by changes in the alveolar walls.

A pathological absorption of the roots of the permanent teeth occasionally occurs, though at rare intervals. I have seen two cases in which practically all of the teeth of the persons were lost in middle life from absorption of their roots. In neither of these cases were there other symptoms than the loosening of the teeth.

Cases in which local absorptions in the roots of the permanent teeth occur are not so very rare. Any one who is extracting many teeth will soon be rewarded by finding some of these

absorptions, if he will carefully clean the teeth. Some will be broad, others small areas of not considerable depth. Others will be very small in area, but deep, looking as much like worm holes as can be imagined. Some of these little holes may reach the pulp canal. Occasionally an absorption reaches the pulp chamber of a molar tooth. It is comparatively rare that these absorptions cause pain. I have, however, seen a few cases in which absorptions invading the pulp chamber did give great pain, which was referred so indistinctly and to so many localities that it was by mere chance that I found them.

While these absorptions occurring within the substance of the root of the tooth may be repaired, so long as they remain covered by the peridental membrane, and are not infected, it does not follow that this may occur if the membrane has been loosened by suppuration, or even if freely exposed to the ingress of the fluids of the mouth by which they become infected. These questions will be considered later.

Attachment of principal fibers of the peridental membrane to the cementum.

The principal fibers of the peridental membrane, which secure the tooth in position, are attached to the cementum by being built around their ends with cementum, and by the calcification of their ends while the cementum is being laid down. This is accomplished by the layer of cells known as cementoblasts, which perform the same function in the building of the cementum that the osteoblasts perform in the building of bone. By some special modes of preparing specimens for microscopic study, these fibers penetrating into the cementum are brought into view. (See Figures 75, 76, 77 and 78.) In many cases it is easy to find areas from which the fibers have been cut away by absorption, and later reattached in a new layer of cementum.

Cementum continuous growing.

The cementum is in a sense a continuous growing tissue. It is always thin on the roots of the child's teeth and becomes thicker as the person grows older; often it becomes very thick in old age. (See Figures 79 and 80.) It is deposited in layers which are similar to the layers of subperiosteal bone, but in the building of cementum these layers are deposited one upon the other, always increasing the thickness of cementum as they are laid down. Each layer represents a new deposit of calcific

material. In this way we may count a large number of layers in the elderly person, but not so many in the young person.

Hypercementosis.

Extraordinary growth of cementum appears frequently, producing what is known as hypercementosis. In this condition the root ends are liable to grow larger and larger by deposits of cementum. Layer after layer may be found in some particular portion of the root, most frequently on the end; or if the roots of two teeth are lying closely together in the bone, they may be united by this extra deposit of cementum. In three or four instances I have seen three teeth with the roots united in this way. (See Figures 81 to 92.)

In sections of such cementum a considerable number of absorption areas that have been filled over with other layers of cementum may generally be seen. In studying cases of growth of cementum we find a considerable deviation from the normal thickness and contour of the tissue, which can only be explained when one understands the nature of the growth; how layer upon layer may be laid down and absorbed again and any part of these absorptions filled in. These frequent absorptions and rebuildings might at first seem to be without order of construction, and yet as we come to understand them we appreciate that they are brought about in a natural and orderly way.

Cementum in animals.

The cementum in the lower animals is similar in most respects to that of man. Physiologically there seems to be no difference. Certain differences in form and thickness are observable. As a rule the cementum of the carnivora, as cats, dogs, etc., is rather thinner than in man.

In the omnivorous animals, and especially in the hog, the cementum is very thick and heavy and is generally well developed. As seen in sections, its layers are generally well arranged, numerous, and the cement corpuscles are large, with as full a complement of fibrils as will be found in the bones. Yet in this splendid development of cementum there is no sign whatever of circulation of red blood. For the best studies of these features an adult animal should be had. If it is growing a bit old, it is still better. (See Figures 76, 77 and 78.)

In the strictly herbivorous animals the thickness of the cementum lies between these extremes and is more like that of man. There is no essential difference in the type of the tissue.

The Peridental Membrane.

ILLUSTRATIONS: FIGURES 93-116.

The term peridental membrane is applied to the soft tissue located between the root of the tooth and the bony walls of its alveolus, or socket. These membranes serve to attach the teeth to the bones of the jaws.

The peridental membrane is a very active tissue, having a rich vascular system and a rich supply of nerves. It is subject to inflammations and suppurations as these occur in other soft tissues. It is able to repair injuries to its own tissue, but does not rebuild its own tissue when any considerable part of this is detached from the cementum by suppuration. If the peridental membrane is cut from the tooth as closely as possible with a sharp lancet, as may readily be done in that part above the crest of the alveolar process, apparently perfect healing occurs very rapidly, provided there is no infection. The tendency of the tissue to come into apposition when cut, as has been mentioned, is a powerful influence for the prevention of infection in such cuts.

In order to test this proposition, I at one time tried the plan of cutting the membrane in this way for depletion in cases of threatened apical pericementitis. In nearly one hundred cases, there were but two that did not heal by first intention. They seemed to be completely restored in one or two days. The two cases were infected and suppuration occurred, which left permanent injuries in the form of shortened gingivæ. This occurred in spite of my care to clean the parts well before making these cuts.

Fibers of the peridental membrane.

The peridental membrane has a special arrangement of fibers, called the *principal fibers,* which are attached to the tooth on the one side, and to the alveolar process on the other. (See Figures 99, 100 and 101.) There are other groups of these fibers; the free gingivæ group, which extend occlusally into the free gingivæ; the trans-septal group, which pass from tooth to tooth over the crests of the alveolar septi; and the alveolar crest group, which pass outward from the cementum and are attached into the crest of the alveolar process. These latter groups were described in our consideration of the gingivæ. Within the peri-

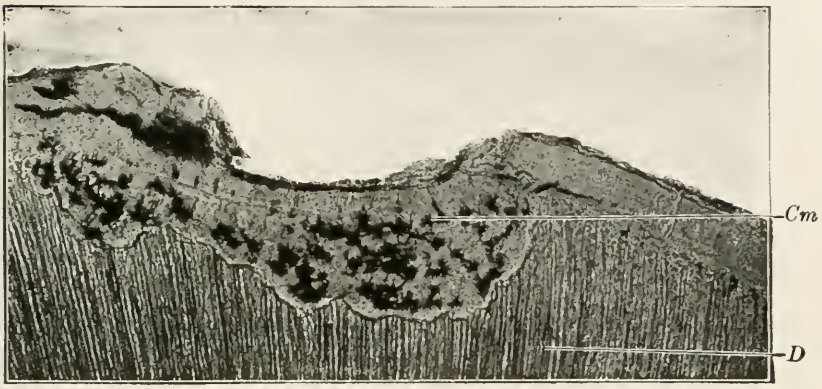

Fig. 68.

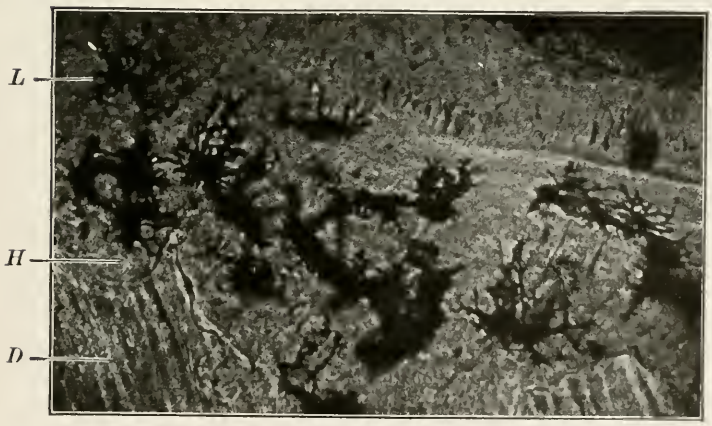

Fig. 69.

Fig. 68. Record in the calcified tissue of an absorption repaired: D, Dentin. Cm, Cementum filling absorption cavity. *Noyes.*

Fig. 69. Thick lamellæ of cementum with many lacunæ, filling an absorption in dentin: L, Lacunæ. H, Howship's lacunæ filled. D, Dentin. *Noyes.*

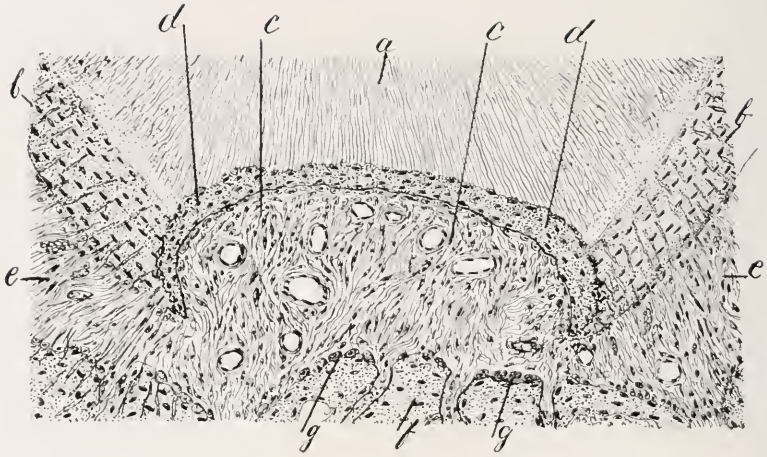

FIG. 70.

FIG. 70. Cross section of the root of a temporary incisor tooth of the pig, showing a large area of absorption which is partly filled in with cementum.

a, Dentin. b, b, Cementum. c, c, Area of absorption. It will be noticed that in this area all of the cementum and a considerable portion of the dentin have been removed. d, d, Cementum that has been laid down upon the surface of the dentin and cementum alike. e, e, Peridental membrane. f, Portion of bone forming the wall of the alveolus that has grown forward into the area of absorption. g, g, Osteoclasts which are removing these bony projections. The bone which has been advanced here to take the place of the absorbed area is being removed again in compliance with the rebuilding of the cementum, which is in progress.

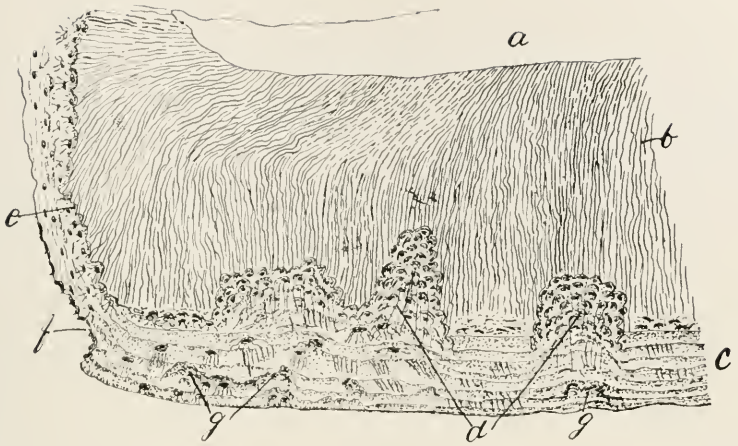

FIG. 71.

FIG. 72.

FIG. 71. One-half of the apex of the root of a lower molar. From a dry section. a, Pulp canal. b, Dentin. c, Cementum. A number of absorptions have occurred at d. Absorptions have proceeded from the second lamella of the cementum and have penetrated the dentin to a considerable depth. These have been refilled with a somewhat irregular deposit of cementum. Along the line e, a very considerable absorption has cut away the entire apex of the root, removing not only the cementum, but evidently a considerable portion of the dentin as well. From the appearance of the incremental lines, this seems to have occurred contemporaneously with those pointed out at d. The exposed dentin has been again covered with cementum, which is fairly regular, though its incremental lines are not clear. f, An absorption that seems to have been in progress at the time of extraction.

FIG. 72. An upper central incisor showing an absorption of a portion of the root. Specimen from Northwestern University Dental Museum.

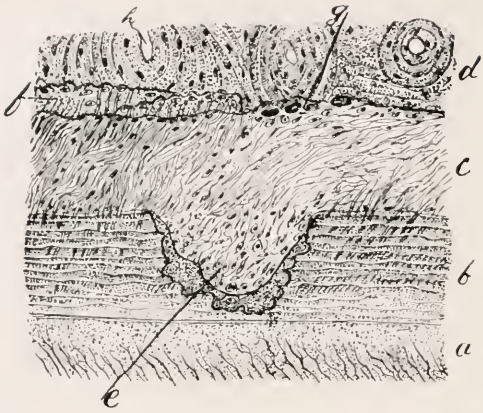

FIG. 73.

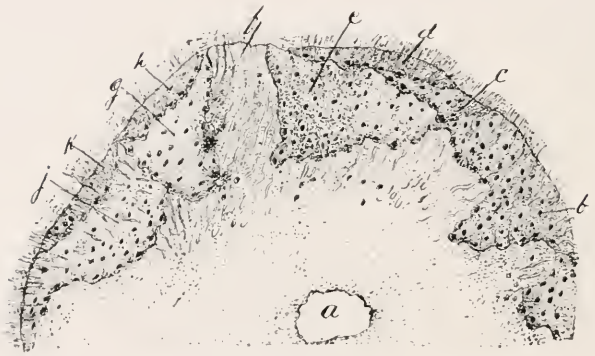

FIG. 74.

FIG. 73. From a section of a bicuspid with its alveolus, showing a pit-like absorption upon the side of the root in which the redeposit of the cementum has begun. a, Dentin. b, Cementum. c, Peridental membrane. d, Bone forming the wall of the alveolus. e, Absorbed area of cementum. It will be noticed that a new deposit of cementum has begun the filling of the area, and that the soft tissue in the area of absorption is of a cellular type. The bone also shows the effects of absorption in the cutting away of portions of the rings of the Haversian systems at f, while at g the presence of osteoclasts shows that absorption is in progress at that point.

FIG. 74. Cross section of the immediate apex of the root of a cuspid tooth, showing large areas of absorption. a, Root canal. b, e, g, and j show extensive absorption areas that have been refilled with cementum, while c, d, h, and k show smaller absorption areas that have occurred later. Some of these areas show the included fibers of the peridental membrane plainly, while others do not, probably for the reason that the section is not parallel with them. At f, the original or regular deposit of cementum reaches the present surface. The plane of the section is not such as to show the incremental lines, and therefore the relation of the absorptions to these can not be seen.

dental membrane proper there are three groups of fibers which deserve particular mention, as follows:

The horizontal group, consisting of those fibers which pass out at right angles to the long axis of the tooth, and are attached to the bone of the alveolar process a little below the crest.

The oblique group, consisting of those fibers which pass from the cementum in an oblique direction occlusally, and are attached to the bone of the alveolar process. These constitute the body of the peridental membrane, or the fibers which cover the main body of the root portion of the tooth.

The apical group, consisting of those fibers which are attached about the apical portion of the root and extend in fan-shaped bundles to the surrounding alveolar process.

It is the function of these three groups of fibers, assisted in some degree by the groups heretofore described, to maintain the tooth in its socket and support it against the stress of mastication. In writing of these groups, I shall continually speak of the fibers as arising from the cementum and being attached to the bone. We might just as consistently speak of them as arising from the bone and being attached to the cementum.

The horizontal group. The fibers of this group are placed close to the crest of the bony alveolar process and pass directly from the cementum to the bone and are attached to it. This is not a broad band of fibers, but varies considerably in different specimens. Sometimes, in the incisor, cuspid and bicuspid regions, we will find this band of fibers about as broad as one-half the thickness of the tooth. The fibers are comparatively short, but very strong. The demarcation between this group of fibers and the alveolar crest group is not at all definite, but the fibers of this group pass directly across and are inserted in the wall of the alveolar process near the crest, while those of the alveolar crest group are inserted into the margin or crest of the alveolar process and have a somewhat curved direction. It seems to be the function of the horizontal group to sustain the tooth against sudden lateral pressure, which may occur in the chewing of food. It is, therefore, a very important group of fibers. This group is very materially assisted in sustaining the tooth against lateral pressure by those fibers of the apical group which pass in the horizontal direction, and tend to prevent lateral motion of the apex of the root. (See Figures 93, 94 and 95.)

The oblique group. The fibers of this group constitute the body of the peridental membrane. Just apically of the horizontal group there is an almost sudden change in the direction of

fibers. They pass across in an oblique direction occlusally from the cementum to the bone. These fibers vary much in their length and some are much more oblique than others. They are gathered into bundles, more or less; some of the bundles are short, passing almost directly across, while others are long, passing quite a distance occlusally within the peridental membrane before they reach the bone.

This description applies to the peridental membrane in a young person or young animal. As the person or animal grows older, and, particularly, when the teeth have been subjected to very severe use, the peridental membrane will become very much thinner and the blood vessels will come to lie in grooves in the alveolar process, so that the spaces between the blood vessels are fully studded with the fibers of the peridental membrane throughout this body portion of the bone. Such teeth are unusually firm in their sockets and will bear great pressure without injury.

Many complete bundles of fibers can be traced from the cementum to the bone; many of them can not be so traced, the fibers seem to split up into finer fibers and re-collect into bundles for insertion into the bone. This arrangement is maintained, with considerable variation, however, over the body of the root of the tooth.

The blood vessels, which pass from the position of the apex of the root, through the peridental membrane in the occlusal direction, lie almost centrally between the root and alveolar process in young subjects. These very much disturb the apparent direction of the fibers. In many microscopic sections the membrane has the appearance of being double, one portion being attached to the cementum of the tooth, the other portion to the bone, divided by the blood vessels. The presence of the blood vessels throws the fibers out of the field in the particular part of the section, but wherever we can cut a section through the length of these fibers, undisturbed by the passage of blood vessels, the idea that the membrane is double is completely dispelled. We are able, then, to trace the fibers from the cementum to the bone, even though they are very long. (See Figures 94 and 95.)

The function of these fibers is to swing the tooth in its socket and sustain it against pressure coming on the occlusal surface or the incisal edge of the tooth. These fibers, about many molar roots, are sufficiently strong to maintain the tooth against three

hundred or more pounds of pressure, which gives an appreciation of their combined strength in supporting the teeth. Bicuspid and molar teeth which are unable to sustain a stress of a hundred pounds or more are lamed in many of the acts of chewing food.

In Northwestern University Dental School we have occupied an hour or more during each year, for a number of years, in demonstrating the strength of the bite, by asking members of the class to make trial bites on a gnathodynamometer. (See Figure 102.) In these trials, we find men in every class who can register three hundred pounds, and some of them appear to very easily register from three hundred and twenty-five to three hundred and fifty pounds.

The apical group. The fibers of this group spread around the immediate apex of the root, standing out fan-like from every part and passing across to be inserted into the bone. These fibers are usually gathered into very definite bundles, and while the fan-like arrangement is correct, as a general description, in many cases these bundles diverge from what would be a true fan-like arrangement to one side or to the other, producing a very considerable irregularity in the crossing of the bundles one over the other to reach the bone. As we approach this fan-like arrangement of fibers around the apex of the root, we find the oblique fibers changing their direction and running more and more directly across the cementum to the bone until they meet the fan-like arrangement of the apical fibers. (See Figures 96 and 101.)

In this position, we have in effect an apical horizontal band of fibers encircling the root, made up of those oblique fibers which are very nearly horizontal and a considerable number of fibers of the apical group which pass outward horizontally or nearly so. While the root of the tooth is swung in its socket by the oblique fibers, these horizontal fibers, which pass across in the change to the fan-like fibers, around the apex of the root, seem to have a special function of holding the apical portion of the root centrally in its socket, against any lateral pressure that may be brought upon the tooth.

The bundle formation of the fan-like fibers, surrounding the apex of the root, gives space to a considerable indefinite connective tissue, which is the seat of the inflammatory condition in the beginning of alveolar abscess.

THE INDEFINITE CONNECTIVE TISSUE.

There is in the peridental membrane, mixed through among the principal fibers, a considerable amount of indefinite connective tissue, forming fibers which usually run somewhat nearly parallel with the length of the tooth, and yet there are a good many exceptions to this rule. They may take almost any course. These are attached to connective tissue cells and they are processes from these cells rather than true fibers. This connective tissue is largely the supporting tissue of the blood vessels, nerves, veins, etc., which pass to and fro in the peridental membrane. It fills up all the interstices between the bundles of the principal fibers. This tissue often disappears almost entirely in old subjects. (See Figures 103 and 104.)

BLOOD VESSELS.

The blood vessels are usually seen as coming into the peridental membrane in the apical space, and splitting up there and running parallel with the length of the tooth to the crest of the alveolus around the tooth, and there connecting with the blood vessels from the gums and the gingivæ. These blood vessels break up into more or less arterial and capillary groupings, as they pass through the peridental membrane. The number of these varies greatly. In injecting specimens of the peridental membrane of animals, we will often find entering the apical space an artery which breaks up into from four to six smaller arteries, and in one case I observed eight branches, each taking its way along the side of the root. Generally, however, there are fewer branches given off in the apical space, and as they take their way into the body of the peridental membrane, they separate into a number of smaller branches. It is sometimes difficult to say whether the blood vessels enter the apical space or terminate at the apical space, because they appear to also enter over the crest of the alveolar process. Blood vessels also enter and pass out from the peridental membrane all about the body of the root through the alveolar process in quite plentiful numbers, each passing through the Haversian canals into the bone and splitting up, more or less, while passing through the bone, giving the bone of the alveolar process a very rich supply of blood, as well as a rich collateral circulation for the peridental membrane. (See Figures 98, 104 and 109.)

NERVES.

The nerves of the peridental membrane follow the same course as the blood supply, and usually these lie in close associa-

tion with the blood vessels which pass through the membrane longitudinally. These also pass through the Haversian canals. A tooth does not lose its blood supply, nor its nerve supply, if the whole of the gingivæ is cut away to the crest of its alveolar process, nor if the whole of the apical space is cleared of tissue at the same time. The collateral circulation through the bone itself is always sufficient to maintain its vitality, and the nerve supply similarly received is sufficient to maintain its sensations. The sense of touch will remain unimpaired, and the sense of pain, in cases of inflammation of the membrane, will be the same as if the peridental membrane were perfect in all of its parts.

It is in this body that the sense of touch of the tooth resides, the slightest touch being registered as such on the sensorium. It will be seen then, that while the peridental membrane is the organ of touch for the root, this sense of touch is not disturbed by the cutting away of the tissues from the gingival end of the membrane, or the cutting away of the tissues from the apical end of the root, or by cutting away both at once. The membrane is found to retain its sensory functions both as to pain and touch, through all this mutilation. The peridental membrane, as the true organ of touch of the tooth, retains this sense as long as there is any of the peridental membrane attached to the bone and to the tooth.

Osteoblasts.

The osteoblasts of the peridental membrane are in no wise different from the osteoblasts of other bone regions in the body. They lie upon the bone of the inner surface of the alveolar process the same as upon the surface of other bone. They almost completely line that surface of the bone next to the peridental membrane, leaving only room for the attachment of the fibers of the peridental membrane, which pass between them. (See Figures 105, 106 and 107.) Whenever the fibers of the peridental membrane lose their attachment to the bone, whether it be by absorption of the bone or otherwise, these cells build on more bone about the fibers and the fiber ends calcify with this bone and are thus reattached to the bony wall. This function occurs in any case where the tooth is moved because of the extraction of a neighboring tooth, or because of the gross movement of the tooth in the jaw. All of these movements call for absorption of bone from one side of the root to give way for the movement, and the fibers are loosened for the time and then again connected in the way mentioned above. Sections through the bone show

that during these movements the fibers are loosened in patches here and there, so that the tooth does not become loose by any wholesale detachment of the fibers. This is a function that is shown in many ways by the movements of the teeth and should be recognized by the dentist.

CEMENTOBLASTS.

The cementoblasts lie upon the cementum of the tooth as thickly as the osteoblasts upon the bony wall of the socket and perform the same function for the cementum as the osteoblasts perform for bone. They are instrumental in the laying down of the calcium salts in the building of cementum, and certain of their number are left in the cementum as living cells, which we call cement corpuscles, the same as osteoblasts are left in the bone as bone corpuscles. (See Figures 108, 109, 110 and 111.)

While all of this is true in a general sense, there is the difference already mentioned that the bone has in itself the function of repair of absorptions, because it is furnished with a circulation of red blood, which penetrates in its influence to every part of the bone tissue, while the cementum has no blood vascular system and has no power of self-repair. So far as has ever been demonstrated, it receives no sustenance whatever through the dentin. All such repairs and all the laying down of the new cementum is done by the cementoblasts of the peridental membrane. Sometimes these absorptions of cementum cut away its entire thickness, and even cut into the dentin to various depths. Wherever we find these repaired, which we do frequently, we find that the whole of the repair is made by cementum which has been laid down by the peridental membrane. (See Figures 66 to 74.) Such repairs are made only under conditions of complete asepsis. In cases in which the peridental membrane has been stripped from the cementum, and the part has become infected and pus soaked, no reattachment whatever may be made to it by living tissue. It is a dead tissue. This is not a new thought, but a very old one. Lister, in one of his articles in 1867, which was before the firm establishment of the influence of micro-organisms in the production of disease, called attention to the fact *"that the mere contact of a foreign body does not of itself stimulate granulations to suppurate; whereas the presence of decomposing organic matter does."* He states that a piece of dead bone "free from decomposition" not only fails to cause suppuration of the surrounding tissue, but may be absorbed; while pus-soaked dead bone always produces suppu-

ration. This quotation is given in full in this book in the consideration of the condition of the cementum in chronic alveolar abscess.

This quotation shows explicitly that the failure of attachment to bone that was dead and pus-soaked was well known long ago, and also the fact that a clean piece of bone planted in the tissue might be absorbed, was at the same time perfectly known. The one of these was as completely dead as the other, but it was the previous saturation of the one by the products of suppuration which prevented it from being absorbed and caused it to keep up the pus formation; while the other, which was not infected, was absorbed by the tissue. So we find the fact to be with regard to cementum. When the tissues have lost their attachment to the cementum, because of pus formation, and the cementum has absorbed this pus into its own tissue, no attachment of living tissue can be made to it, as has been mentioned.

Examination of the soft tissue overlying an area of such detachment reveals the additional fact that these specialized cells, the cementoblasts, the function of which is to build cementum, have disappeared, evidently having been destroyed by the suppurative process which caused the detachment. As will be shown later, the principal fibers of the peridental membrane, which were attached to the cementum over such an area, will also have disappeared, together with the corresponding portion of the bone of the alveolar process to which they were inserted. We, therefore, have not only those conditions which make a reattachment impossible, but the cells which would under favorable conditions make such an attachment, have disappeared, as have also the fibers which would be attached.

These are among the most important facts in dental pathology and, unfortunately, have not been heretofore recognized by many dentists.

Epithelium.

Among the tissues of the peridental membrane there is a plentiful distribution of epithelial cells. These lie for the most part close to the cementum but never touch it. They are mingled among the principal fibers, generally in the form of strings, sometimes clubbing together so as to be several cells thick, but often in strings of single cells touching each other. These occasionally form loops which extend outward into the substance of the membrane for one-half its thickness, or more, and then dip

back again to a position near the cementum. These cells are found everywhere around the root of the tooth, but in general the strings are so disposed as to run lengthwise of the root. (See Figures 112 to 116.)

I was very much puzzled when I first observed these cells. They had the appearance of epithelium and stained like epithelium, but I could not conceive at the time the possibility of epithelial cells growing in such a position. These cells were discovered during my investigation of the periosteum and peridental membrane, and in writing on this subject in 1886-7, I called these strings of cells lymphatics. I later realized that they really were epithelial cells, and corrected the error in naming them. I know of no other occurrence in the body, of epithelium among the connective tissues in any similar form. In fact, the whole appearance of the cellular forms seems to be out of place and accidental, and yet they are found in all the animals of the higher type, as well as in man, and therefore can not be regarded as accidental. They belong to the tissues of the peridental membrane, but their function has been a question for many years.

It has been claimed by some recent writers that these strings of epithelial cells are the remains of the breaking up of the enamel organ; the cells floating away and taking these irregular forms. To my mind this could not be. I have followed the breaking up of the cells of the enamel organ. I have seen them float away into the tissues in groups forming epithelial pearls, some large and some very small. I have followed them from one age of an animal to another age, and as I have thus followed them I have found that they were absorbed and disappeared completely. These cells seem to have nothing whatever in common with the epithelial cells scattered in strings in the peridental membrane. They are not like them. They do not seem to be of the same quality at all.

Recent investigations of these cells by German histologists have been carefully reviewed by Dr. Th. Dependorf,* and his review has been translated and printed in the Northwestern Dental Journal.† Some of the writers seem to have done close microscopic work, and while they differ on many questions regarding these groups of cells, there is one upon which there is

* Zur Pathogenese der Zahnwurzelzystem. Deutsche Monatsschrift für Zahnheilkunde, 1912, p. 809.

† Dr. Anna A. Oppermann, Northwestern Dental Journal, Vol. X, p. 73; Vol. XI, p. 9.

Fig. 75.

Fig. 75. Two fields of cementum showing penetrating fibers: Gt, Granular layer of Tomes. C, Cementum not showing fibers. F, Penetrating fibers. *Noyes.*

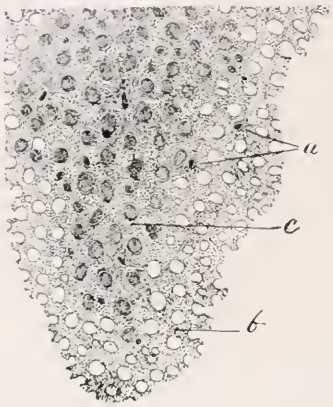

FIG. 76.

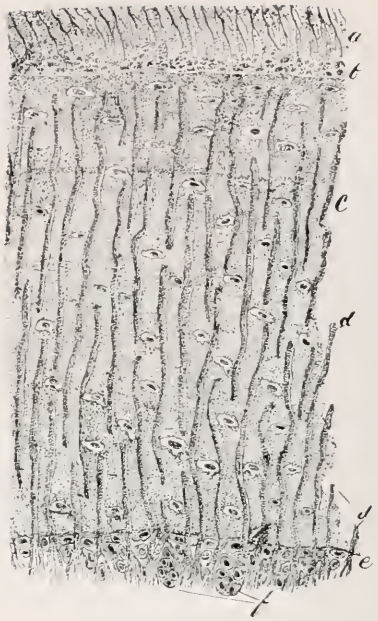

FIG. 77.

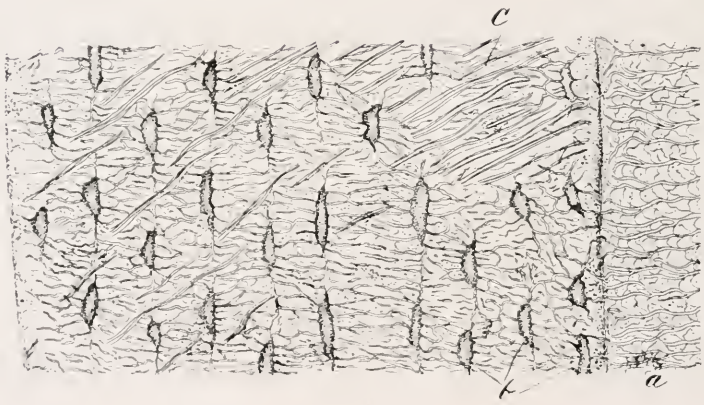

FIG. 78.

FIG. 76. Section of cementum of pig cut horizontal to and near the surface of the root of the tooth showing cross sections of the included fibers. b, Thin margin of section, from which the fibers have fallen out of their alveoli. c, A little thicker portion in which the fibers remain. It will be noticed that from shrinkage the fiber is a little small for its alveolus, so that it is slightly separated from one side. a, Cement corpuscles.

FIG. 77. Longitudinal section of the cementum of a pig, showing the included fibers of the peridental membrane. e, Margin of cementum showing fibers passing from the cementum to the peridental membrane, and the layer of cementoblasts with other cells in the neighborhood. f, Epithelial cells. d, d, Fibers protruding from broken margin of section. a, Dentin. b, Junction of dentin and cementum.

FIG. 78. Cementum of pig from the dried section. a, Dentin. b, Lacunæ of cementum with canals anastomosing with each other. c, Imperfectly calcified fibers. It will be noticed that a few of the dentinal tubes pass through into the cementum.

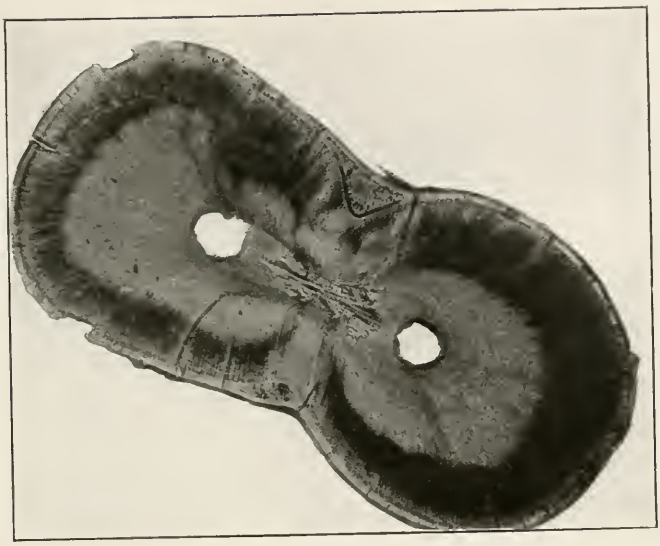

FIG. 79.

FIG. 80.

FIG. 79. A transverse section of a root extracted from a young person. The cementum is thin, but is thicker in the groves on the proximal sides. *Noyes.*

FIG. 80. A transverse section of a root from an old person. This root had carried a crown for many years. The section was cracked and one edge broken. *Noyes.*

Fig. 81.

Fig. 82.

Fig. 81. Hypertrophy of the cementum on the side of the root of a lower molar near the gingival line of the tooth. From a longitudinal section, man. a, Dentin. b, Cementum. c, Fibers of peridental membrane. From b to c the cementum is normal, and the incremental lines fairly regular, but at d, one of the lamellæ is greatly thickened. At e, this lamella is seen to be about equal in thickness with the others.

The next two lamellæ are thin over the greatest prominence, but one is much thickened at g and both at h. These latter seem to partially fill the valleys which were occasioned by the first irregular growth.

Fig. 82. Hypertrophy from root of cuspid, man, in which the irregularity is confined to the first lamella. a, Dentin. b, Thickened first lamella. c, Subsequent lamellæ, which are seen to be fairly regular.

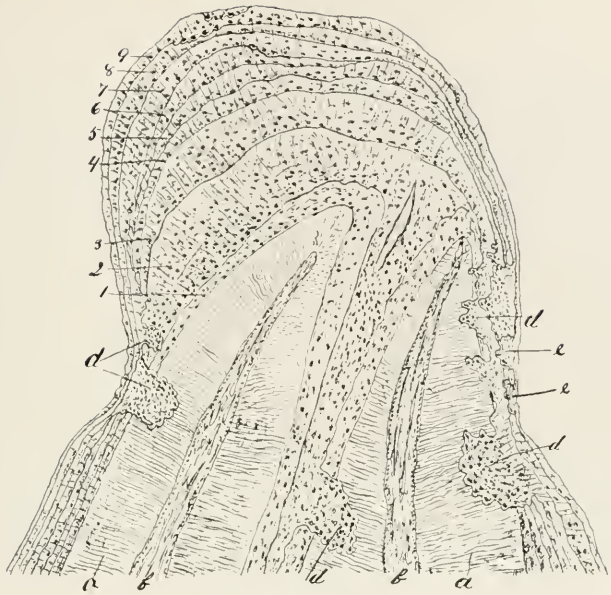

FIG. 83.

FIG. 84. FIG. 85. FIG. 86. FIG. 87.

FIG. 83. Apex of root of an upper first bicuspid tooth with irregularly developed cementum. a, a, Dentin. b, b, Pulp canals. The lamellæ of cementum are marked 1, 2, 3, etc. d, d, d, Absorption areas that have been refilled with cementum.

It will be seen that the apices of the roots were originally separate, but became fused with the deposit of the second lamella of cementum, and that in this the irregular growth began and was most pronounced. It has continued through the subsequent lamellæ, but in less degree. It will also be noticed that the absorption areas, d, d, d, have proceeded from certain lamellæ. Between the roots this has broken through the first lamella and penetrated the dentin, and has been filled with the deposit of a second lamella. Other of the absorptions have proceeded from lamellæ, which can be readily made out. The small points, e, seem to have been filled with the deposit of the last layer of the cementum, while others have one, two or more layers covering them.

FIGS. 84, 85, 86 AND 87. Teeth with extensive hypercementosis. Specimens from Northwestern University Dental Museum. Tooth shown in Fig. 84 presented by Dr. T. A. Black, Galveston, Texas. Teeth shown in Figures 85 and 86 presented by Dr. Amy Bowman, Los Angeles, California.

FIG. 88. FIG. 89. FIG. 90.

FIG. 91. FIG. 92.

FIGS. 88, 89 AND 90. Teeth with roots fused by the coalescence of cementum. Specimens from Northwestern University Dental Museum.

FIGS. 91 AND 92. Teeth with roots fused by the coalescence of cementum. Specimens from Northwestern University Dental Museum. Teeth shown in Figure 92 presented by Dr. A. S. Cheeseman, Joliet, Illinois.

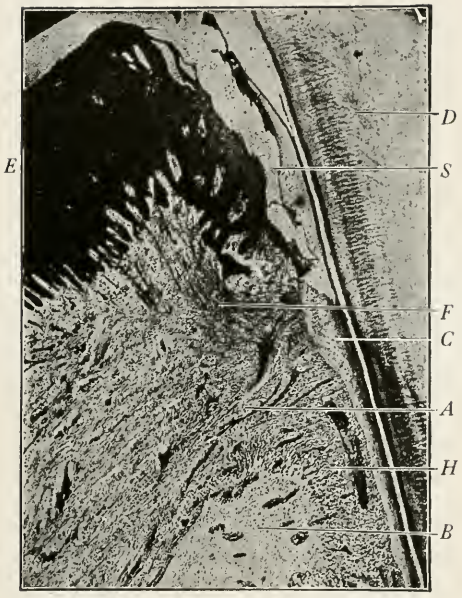

Fig. 93.

Fig. 93. Longitudinal bucco-lingual section through root of tooth and gingival portion of investing tissue showing fibers of the peridental membrane. E, Epithelium. D, Dentin. C, Cementum. S, Subgingival space. F, Free gingivæ group of fibers. A, Alveolar crest group of fibers. H, Horizontal group of fibers. B, Bone of alveolar process. *Noyes.*

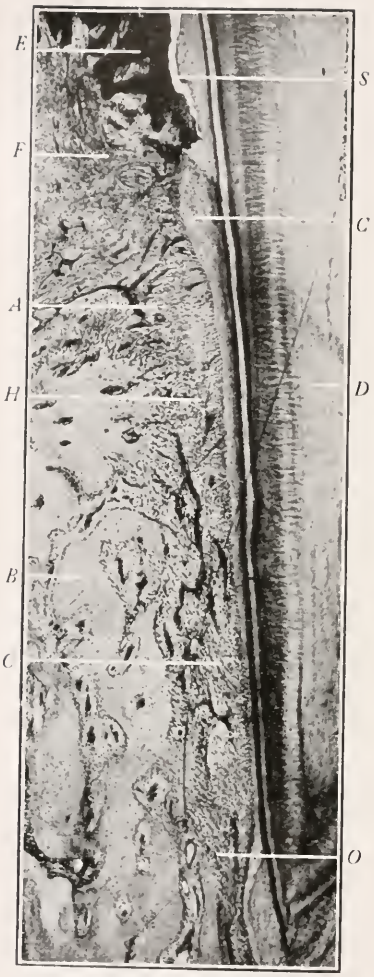

FIG. 94.

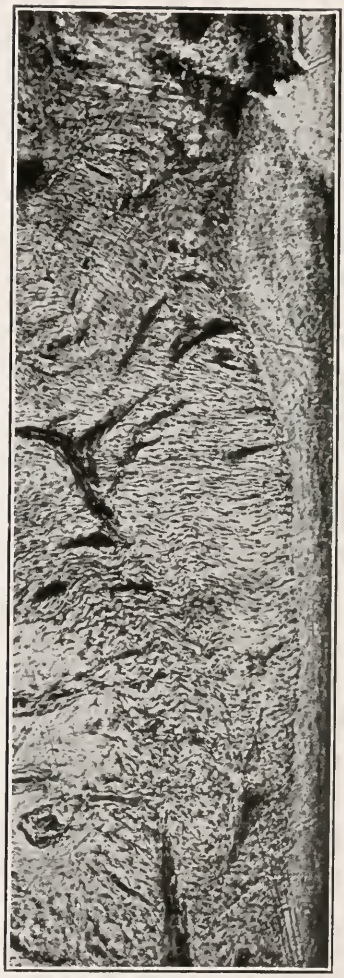

FIG. 95.

FIG. 94. Longitudinal section, slightly oblique, through root and peridental membrane, showing fibers of the peridental membrane. E, Epithelium. D, Dentin. C, Cementum. S, Subgingival space. F, Free gingivæ group of fibers. A, Alveolar crest group of fibers. H, Horizontal group of fibers. O, O, Oblique group of fibers. B, Bone of alveolar process.

FIG. 95. A higher magnification of a part of Figure 94.

general agreement, viz., that these strings of epithelial cells multiply in the peridental membrane, break up, form sheets of cells which surround certain areas of infection, and cut them off by encysting them. The activities of these cells will be more fully considered under the subject of cyst formation.

Physiological Powers of the Peridental Membrane and Cementum, as Shown by Planted Teeth.

ILLUSTRATIONS: FIGURES 117-119.

The physiological powers of the several elements which compose the peridental membrane, their correlation to the cementum in minute anatomy and in function, and especially their power and lack of power to repair themselves and renew their physiological connections with their related tissues when injured by disease or accident, are of much more than usual importance. Dentists have been very slow in gaining an understanding of the peridental membranes and their relations to the tissues with which they are connected, because they have made so little practical study of these tissues. Really they have had but little opportunity to know this subject, since it has been taught very superficially, if at all, in dental schools. In efforts made by the general profession to study the diseases of the peridental membrane and cementum, they have disregarded the powers of these tissues, and therefore have failed to gain the most beneficial information.

The planting of teeth.

The famous surgeon, John Hunter, whose professional activities were around 1750-90, and who seems to have given unusual attention to the teeth, did quite a little in transplanting and implanting teeth. This operation seems to have become quite popular in Europe during the latter part of the century, but soon died away and was lost to sight. It was not, however, original with Hunter. Guerini gives credit to Amboise Parè for having first performed and described this operation. It had been known and practiced and had passed out of use several times before his day. Even after the beginning of the last century, the transplantation of human teeth was practiced in France, and perhaps elsewhere. There was developed a practice of transplanting directly from one human mouth to another. The dentist, being applied to for a new tooth, would, after determining what was needed, find some person who was willing to

sell a similar tooth for a price. Or a good careful lady would bring with her a servant girl who would sell one of her teeth. Then the carious tooth would be extracted from the patient and thrown aside. The similar tooth was then removed from the mouth of the person furnishing the tooth for replacement. The tooth was at once transferred to the alveolus, from which the carious tooth had been removed. (See Figure 117.)

The practice seems not to have lived very long, nor to have become very general. No careful records of operations of this character seem to have been kept. Neither can we suppose from the writings which have come down to us that the powers of repair in the tissues involved were given careful study. I have been over most of the old writings in the English, German and French languages, and I do not now remember of a single case in which there was an effort to make a close study of this membrane, or of its physiological powers.

During the century just passed there were occasional revivals of the practice of planting teeth in the sockets from which teeth had been extracted, often using old dried teeth for the purpose. These efforts were usually individual in character in that they were confined to a few persons.

In more recent years, the practice has been revived on the assumption that by the use of antiseptics, lasting operations both in transplantation and implantation might be made. A good many operations were made and attracted considerable attention at dental society meetings, but as time wore on it was found that these operations failed after a few years, the same as others had done.

However, with the more careful following of cases which had become possible, the advisability of this operation in individual cases was better studied. The result has been that a few operators have made the most possible of its use in carefully selected cases. So far as I have knowledge, transplantations from person to person have not been made in recent years, except by a very few practitioners.

During the revival of this procedure, a nomenclature became fairly established representing the different classes of planting operations.

Replantation is used when teeth are extracted purposely or by accident, and replaced in their own sockets.

Transplantation is used when a stranger tooth is placed in the socket of a tooth just removed.

Implantation is used when the teeth have been removed at some former time and a new socket is cut in the residual alveolar process, or ridge, and a stranger tooth planted in it.

Taking the mass of evidence afforded by the history of planted teeth, there has been every degree of success and failure which seems possible. A study of these successes and failures illustrates the physiological powers of the tissues to make repairs when the peridental membrane and the cementum have been separated from any cause, or when the wound made by the extraction has healed and all vestige of a peridental membrane has disappeared. The lesson thus learned of the powers of the tissues under such conditions is the all important thing for which this recitation is made.

A considerable number of cases were failures from the start. Suppuration occurred in the socket about the tooth, and it had to be removed within a few days or weeks. It seems to have been sufficiently demonstrated that no attachment would be made in areas of suppuration. Often there will be some suppuration about the gingival margin of otherwise successful cases. My personal observation is that the soft tissue never becomes attached over such areas, and the gum quickly shrinks away, leaving some portion of the root bare. In other cases the soft tissues heal about the teeth, and they become tight or fixed immovably in the sockets. These teeth, which seem to present the most perfect condition, are usually lost within one, two or three years, by absorption of the root. (See Figures 118 and 119.) If such a tooth remains useful for five years, it is regarded as a first-class result.

No HISTO-PATHOLOGICAL STUDIES OF PLANTED TEETH. Nothing is yet very certainly known of the histo-pathology in these cases. A number of the remains of absorbed roots have been cut for microscopic study, but these have given little information. I do not now remember of any case in which the root of the tooth, with its soft tissue and bony investments as it was in the jaw, has been prepared for the microscope and studied by a competent observer. Therefore, we are yet without definite information from the histological standpoint as to what occurs either as the tooth becomes tight, the tissue by which it is held, or the process of absorption by which it is removed.

In reciting this history I have had no intention of describing in detail the several operations of planting teeth. My purpose has been to illustrate a principle in the coördinate physiology controlling the relations of the cementum and the peridental

membranes. Particular note should be made of the fact that in some of these operations, old dried teeth, which had been out of the mouth a long time, have been selected for planting. These seem to have done about as well as comparatively fresh teeth, but not so well as teeth which were removed or displaced by accident, and in which the same tooth was immediately replaced into its own socket.

It has been said that John Hunter, whom I have mentioned, had at one time several roosters about his yard with two or more human teeth planted successfully in their combs. Also that an enthusiastic Frenchman succeeded in planting a human tooth in the forehead of a rat, and it became firmly fixed to the skull. I will not vouch for the correctness of either of these stories, but from that which I have myself seen, I believe both are possible.

Chemotaxis. The meaning of the above line of facts is that there exists between the soft tissue cells, or some of them, and the cementum covering the roots of the teeth, or even the dentin itself, a *positive chemotaxis* which causes the soft tissue cells to seek, or to approach, the cementum, to develop in contact with it and attempt to make an attachment.

The principle in nature expressed by this word chemotaxis (Chemo—Chemistry, and taxis—orderly, or in an orderly way) has assumed great importance within recent years in physiology, pathology, bacteriology and immunity from, or susceptibility to, infections and infectious diseases. It serves to explain many things which previously seemed incomprehensible.

The Standard Dictionary gives the following definition: "Chemotaxis: The property which certain living motil cells possess of approaching (positive chemotaxis) or moving away from (negative chemotaxis) chemical substances of various kinds. Chemotaxis seems to play an important part in some phases of inflammation. Thus it appears to be, in part at least, through the incitement of chemotaxis by the chemical substances which they contain or eliminate, that bacteria act in producing suppuration."

Stedman's Medical Dictionary gives the following definition: "Chemotaxis: Reaction of living protoplasm to a chemical stimulus whereby the cells are attracted (positive chemotaxis) or repelled (negative chemotaxis) by acids, alkalies or other bodies exhibiting chemical properties."

It is through the principle expressed in these definitions that many of the physiological and pathological reactions occurring

between tissues of different kinds are brought about. It seems also that it is a principle which is often active in infective invasions of micro-organisms, or in preventing such invasions, and thus bringing about conditions of susceptibility or of immunity.

On the basis of a positive chemotaxis, we may explain a fact that is sufficiently apparent in the line of experiment recited. When a tooth, which has been extracted for some time, is planted in the tissues of the jaws, whether in a previously existing socket of a tooth or a socket cut for it, under conditions fairly favorable to the healthful action of the soft tissue elements of the neighborhood, there is an immediate tendency for the cellular elements, or certain of them, to attach themselves to the root of the tooth and develop there into fixed tissue. In this way they form an attachment to cementum, or even to dentin. A growth occurs that we may suppose endeavors to reform, or to form an ordinary peridental membrane. It is more probable that the tissues act much in the same way in which open wounds heal by the formation of cicatricial tissue.

As the peridental membrane is a specialized tissue, having in its make-up several kinds of cellular elements and of fibers specially arranged for the performancec of its special functions, we must suppose, from the uniform disastrous results which follow all forms of planting teeth, that at least some parts of these have failed, if indeed there is any definite attempt to reform them. Therefore the connection is physiologically unstable. For this reason the absorptive process begins in the root and continues more or less rapidly until the tooth is cast off.

Attachment of planted teeth physiologically unstable.

The absorption of the root is the universal result of all kinds of planting of teeth in the jaws. The history, therefore, shows conclusively that the attachment is physiologically unstable. It shows further, that positive chemotaxis is changed to negative chemotaxis whenever the root of the tooth, or some portion of it, has been exposed to suppuration and has presumably absorbed products of suppurative decomposition. If we compare the conditions of the peridental membrane and the cementum in cases of replantation and in cases where pockets have formed beside the roots of teeth, we find the conditions so similar that we may well regard them as the same. With the suppuration which has occurred and the loosening of the soft tissues, we must suppose that the integrity of the peridental membrane, as such, and of its special elements which fit it for the performance of its functions,

have been destroyed, and that the tissue in its place is ordinary gingival or gum tissue, which has been modified and weakened by repeated inflammations and suppurations. Over against this is a cementum that has lost its positive chemotactic qualities by having absorbed the products of suppuration. To cap the climax of disabilities present in these cases, they are placed in a field constantly exposed to active infective elements, and are constantly being reinfected. The conditions are such that these reinfections are not preventable.

When we compare the above with a fresh wound created by forming a new socket in the residual alveolar process, or by clearing a socket where a tooth has somewhat recently been extracted, we will see at once that the condition of the tissue in a socket so prepared for the reception of an implantation is in better condition to invite an adhesion of soft tissue than in any case in which the tissue has been detached by an infection alongside a root.

Under all of these adverse conditions, how shall we expect pockets to heal, reattachment of the soft tissues to the cementum to occur, and permanent cures of this condition to be made? As a matter of fact, such cures do not occur, nothwithstanding the reports of successes.

As I must acknowledge that in the past I was for a considerable time deceived by the appearance of betterment which followed treatment, and supposed and stated, as will be found in the article I prepared for the American System of Dentistry, that actual cures occurred under these conditions, the above statement should not be taken as offensive by any one now in practice.

The Alveolar Processes.

ILLUSTRATIONS: FIGURES 120-121.

The alveolar process is the projection of bone which grows up around the roots of the teeth, and forms the sockets in which the roots of the teeth are held by their membranes.

These sockets are the alveoli of the teeth, or if we speak in the singular, each socket is the alveolus of a tooth. The word alveolus means a hole. The alveolar process is the wall of bone around the hole. This is not a separate piece of bone but is continuous without demarcation with the bones which form the maxillæ. There seems to have been no rule among writers on dental subjects as to the use of the singular and plural forms, alveolar process and alveolar processes.

The peridental membrane, as united to the cementum on the one side and to the alveolar wall on the other, connects and binds together the root of the tooth and its alveolar process, thus holding the tooth in position. (See Figures 97 and 98.) Really there is but a single alveolar process in each jaw, which passes around the arch in a single bony projection in which there are the number of alveoli for the accommodation of the roots of the teeth. In most cases the projection of the alveolar process above the body of the bone is not sufficient to accommodate the full length of the roots of the teeth, and the alveoli are sunk into the body of the bone so far as may be necessary.

The alveolar process does not quite cover the gingival portion of the cementum, but stops about two millimeters short of the gingival line of the tooth, different specimens varying somewhat from this measurement. The crest of the alveolar process is therefore always lower than the gingival lines of the teeth.

The alveolar processes are bone.

The alveolar processes are bone, pure and simple, with all of the endowments of the bones in general. Their blood supply is richer than that of most bones, their Haversian canals are larger, and the amount of blood passing through them is greater than in the bones in general. The nerve supply is also richer. To accommodate this very rich circulation, the alveolar processes are permeated by many Haversian canals and a large number of these pass through directly or indirectly from the side of the mucous membrane to the side of the peridental membrane, or the reverse, giving to the peridental membranes a rich collateral circulation through the alveolar wall.

Wherever the bone constituting the alveolar wall is considerably thickened, it has a fairly solid cortical, or surface portion, toward the mucous membrane side, and a thinner, fairly solid portion on the side next to the peridental membrane. In the central portion between these two, the bone is much less dense. Indeed, wherever there is thickness enough to permit it, it becomes cancellous or medullary. It is divided in many directions with thin laminæ of bone, uniting the whole together in a strong mass, in which the interspaces are filled with connective tissue, blood vessels and nerves, giving it a physiological activity closely related to the ordinary connective tissues.

The alveolar process rises much higher above the true form of the maxillary bones in the front part than it does in the back

part of the mouth. It is therefore higher about the incisors and cuspids and lowers away toward the back part of the mouth until, in the lower jaw particularly, the alveoli for the second and third molars are often hollowed out in the body of the bone. Indeed in many cases the alveolar walls on the lingual sides of these teeth are built out around them as they lie one-half, more or less, out on the lingual side of the bone. In the upper jaw a much more decisive alveolar ridge is maintained even to the third molars, but this part of the ridge is much lower than in front.

Development of the alveolar processes.

One who has followed carefully the development of the teeth and the dental arches in the clinical way, together with the occlusion of the teeth, in many children, and the malocclusions which occur among them, will have discovered that the teeth are not made to fit their alveoli, but that the alveoli are made to fit the teeth. The teeth, during the development of the arches, go on with their movements as the bones of the face are growing and expanding from the face of the child to the face of the adult. The teeth are assuming the adult positions by which they are assisting in rounding out the prominences of the adult features. During this time the alveolar processes are keeping even pace with the movements of the teeth. As the teeth move forward, the alveolar walls are absorbed here and built out there, to accommodate the movement. (See Figures 120 and 121.) If some one or more of the teeth are taking wrong positions, bringing about malocclusions, they are not limited or perceptibly held back by their alveoli; but the alveolar walls will be changed in form and built to fit the teeth in the malposition. A cuspid tooth that is crowded forward out of its normal position, for instance, has the walls of its alveolus changed to accommodate this movement. It is not crowded out of its alveolus. It does not lose the fitting of an alveolar wall around it because the tooth has taken a wrong position. This exhibits in part the related physiological factors existing between alveolar processes and teeth and the bones of the face.

All of this goes to show that in the related physiological factors between the positions of the teeth in the arch and the formation of the walls of their alveoli, the teeth are accommodated by the growth of bone about them, and are given the support that the performance of their functions demands. Even if there are supernumerary teeth, not usually reckoned with as normal, their alveolar process is built about them in any position

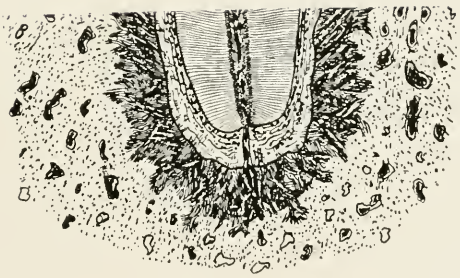

Fig. 96.

Fig. 96. Drawing representing a longitudinal section, to illustrate the fan-shaped fibers. It is almost impossible to get an actual section through the tissues which shows the arrangement, on account of the interlacing of the various bundles of fibers.

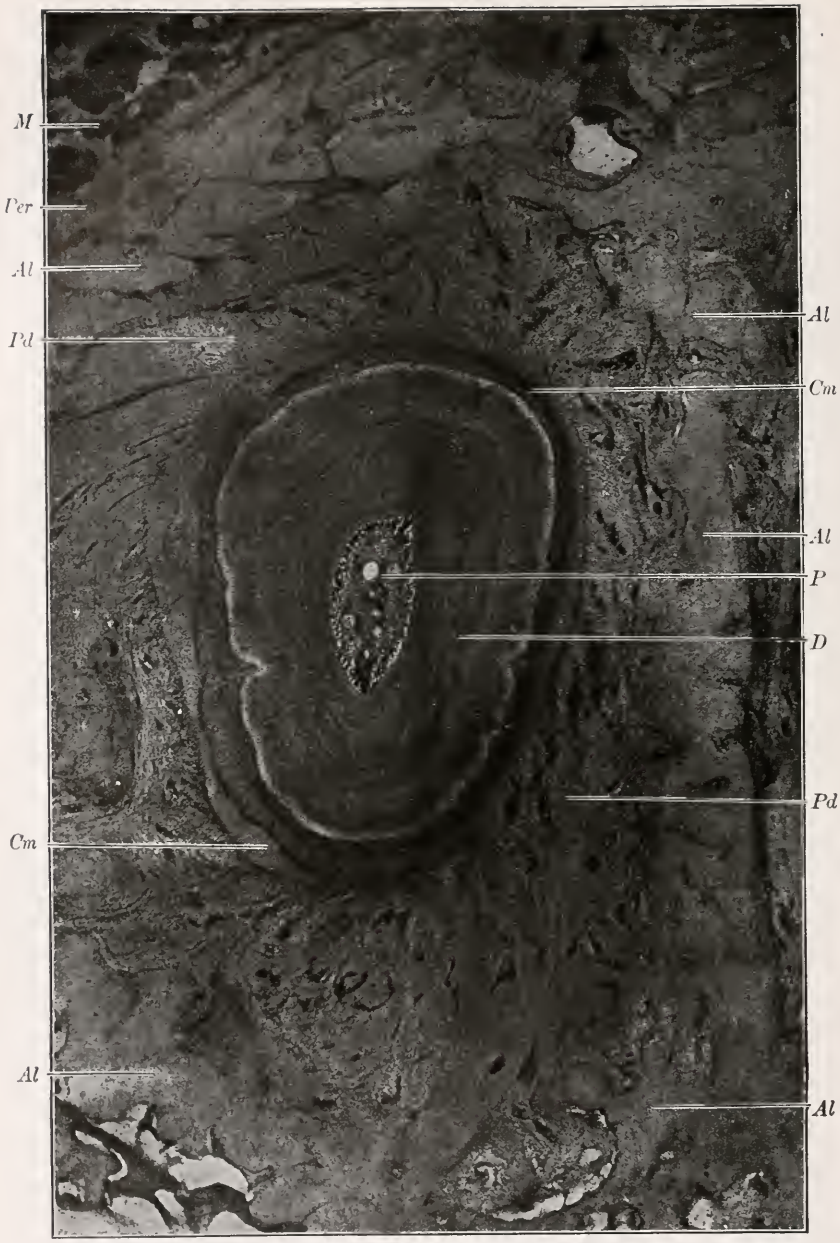

FIG. 97.

FIG. 97. Transverse section of the peridental membrane in the occlusal third of the alveolar portion (from sheep). M, Muscle fibers. Per, Periosteum. Al, Bone of the alveolar process. Pd, Peridental membrane fibers. P, Pulp. D, Dentin. cm, Cementum. *Noyes.*

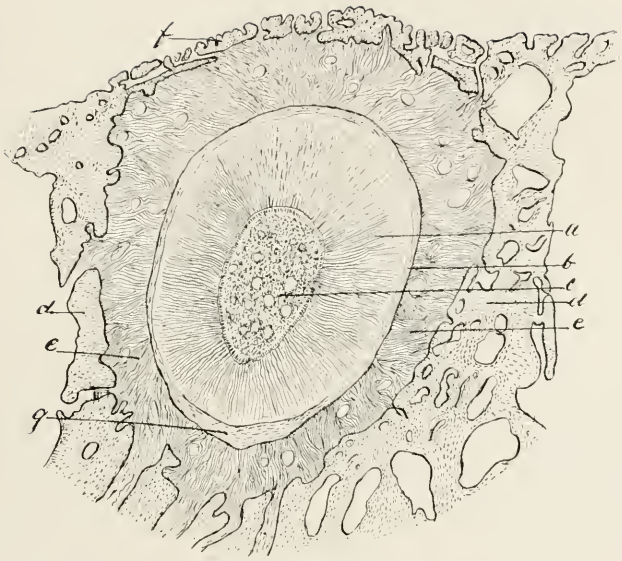

Fig. 98.

Fig. 98. Cross section of the root of a temporary incisor with the peridental membrane and alveolar walls, at about the middle of the lower third of body of the peridental membrane, showing the direction of the fibers of the membrane, and the position of the blood vessels. a, The dentin. b, Cementum. c, Pulp. Its blood vessels are shown. d, d, Alveolar wall, septi between the teeth. e, e, Peridental membrane. The direction and arrangement of its fibers have been carefully represented; also the position and relative size of its blood vessels. f, Thin portion of the anterior alveolar wall. g, Hypertrophy of the cementum.

Fig. 99.

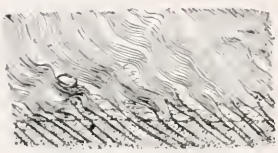

Fig. 100.

Fig. 101.

Fig. 99. Fibers of the peridental membrane passing from the cementum a, to the alveolar wall b. The section is from the root of a first molar of a man about seventy years old. The point chosen for this illustration includes a portion of a strong band of solid fibers c, which pass unbroken from the cementum to the bone. More generally, the fibers, after emerging from the cementum, break up into finer fibers or fasciculi, as at d. This form of the fibers is better shown in Fig. 100.

Fig. 100. Fibers emerging from the cementum and breaking up into fasciculi. From the peridental membrane of a molar of an aged person. This represents the more usual form of the principal fibers, as seen in old age in man. They pursue a somewhat wavy course, and generally the identity of the individual fiber is lost. They are inserted into the bone in compact bundles similar to those of the cementum.

Fig. 101. A group of fibers emerging from the cementum near the apex of a root and radiating fan-like. On either side, the principal fibers are absent for a little space, which is filled with indifferent tissue. From the apical space of a bicuspid of an old person.

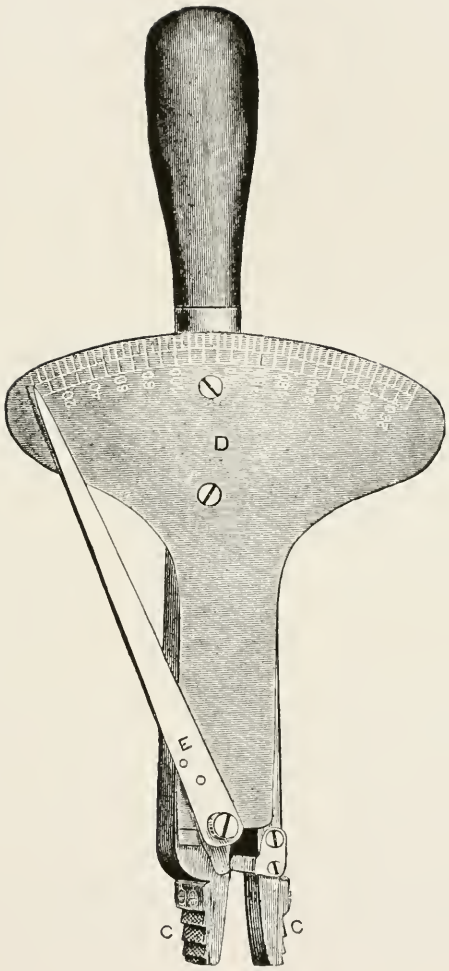

Fig. 102.

Fig. 102. The gnathodynamometer, about two-thirds natural size. Face view. c, c, The rubber pads bitten upon in determining the pressure of the teeth. d, Scale of pounds. e, Needle which marks the pounds. In use this needle remains stationary at the highest point reached until it is moved by the fingers.

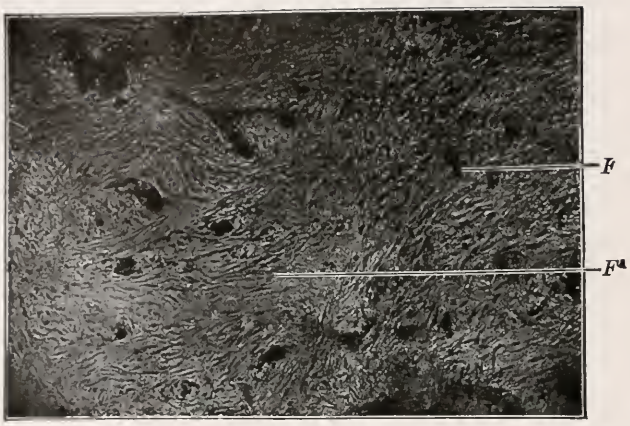

FIG. 103.

FIG. 104.

FIG. 103. Fibers and fibroblasts from transverse section of membrane: F, Fibers cut transversely. F¹, Fibers cut longitudinally, showing fibroblasts. *Noyes.*

FIG. 104. Peridental membrane from perpendicular section of a tooth of the pig, stained with nucleus tinting stain. a, Cementum. b, Bone. c, Blood vessels cut diagonally. d, Nerve bundle. e, Epithelial cells. A number of strings and clusters of these are seen near the cementum. The principal fibers are transparent, while the interfibrous tissue is stained. The cellular elements appear in rows between the principal fibers, which are large and strong near the bone, and only partially break up into fasciculi in the central part of their length.

Fig. 105.

Fig. 105. Penetrating fibers in bone. Pd M, Peridental membrane. ob¹, Osteoblasts of peridental membrane. ob², Osteoblasts of medullary space. Pd B, Solid subperidental and subperiosteal bone with imbedded fibers. Ms, Medullary space formed by absorption of the solid subperidental bone with imbedded fibers. H, B, Haversian system bone without fibers built around the medullary space. *Noyes.*

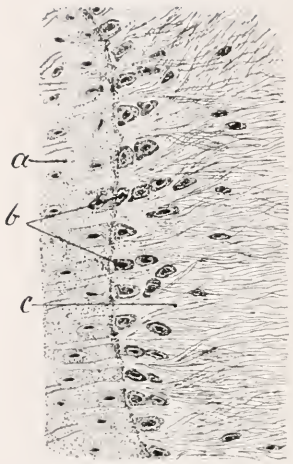

Fig. 106.

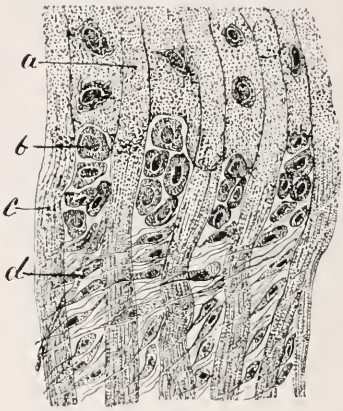

Fig. 107.

Fig. 106. From section including a portion of the alveolar wall, and portions of the peridental membrane, showing the osteoblasts. a, Bone. Inner margin of alveolar wall, showing residual fibers. b, Osteoblasts. Developing cells are seen in the neighborhood. c, Fibers of the peridental membrane. It will be noted that these spring from the bone as solid fibers and immediately break up into fasciculi.

Fig. 107. From section including a portion of the alveolar wall, and fibers of the peridental membrane at a point where these latter are large and compact, and with interfibrous tissue between them. a, Bone showing the large residual fibers. b, Osteoblasts filling spaces between the fibers. c, Principal fibers of peridental membrane, which at this point maintain the solid form far out from the bone. d, Interfibrous tissue consisting of fibroblasts and fibers which lie between the principal fibers and pursue an independent course. Compare with Fig. 106.

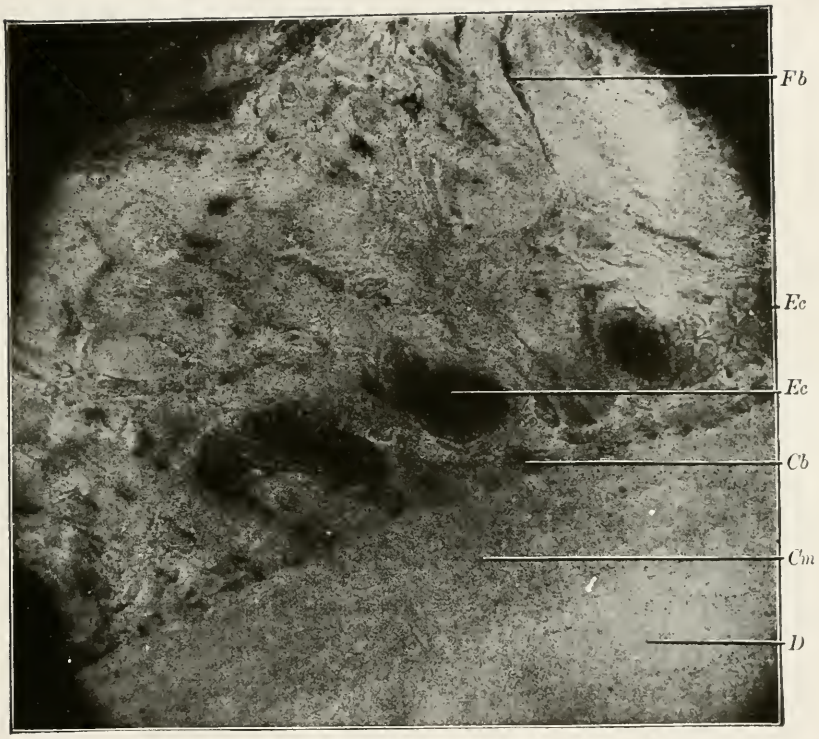

FIG. 108.

FIG. 108. Transverse section, showing the cellular elements. FB, Fibroblasts. EC, Epithelial structures. CB, Cementoblasts. CM, Cementum. D, Dentin. *Noyes.*

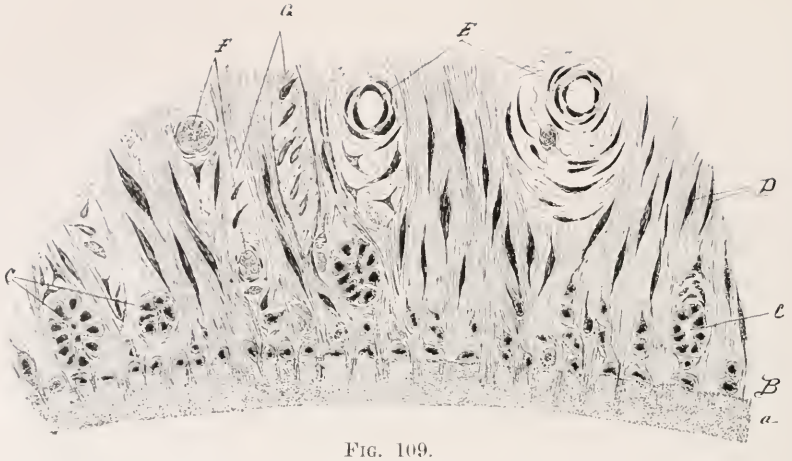

Fig. 109.

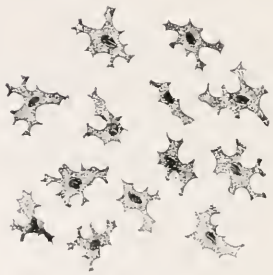

Fig. 110.

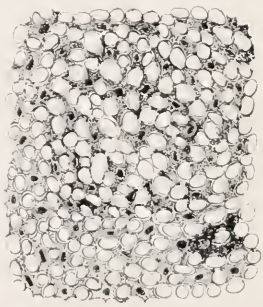

Fig. 111.

Fig. 109. Cementum and portion of the peridental membrane from the sheep. From a cross section of the tooth. A, Cementum. B, Cementoblasts lying between the fibers, which later break up into fasciculi immediately after leaving the cementum. C, E, Cross section of epithelial clusters. D, Fibroblasts. E, Blood vessels. These are accompanied by a large amount of inter-fibrous, or indifferent connective tissue. F, Nerve bundle. G, Fasciculi of fibers pursuing a direction different from the main trend of the principal fibers.

Fig. 110. Cementoblasts isolated to show the peculiar irregular forms of these cells.

Fig. 111. Cementoblasts, in situ, with cross sections of the principal fibers of the peridental membrane of the pig, from a section cut horizontal to the surface of the cementum and including these cells. It will be seen that the cementoblasts fill all the space not occupied by the principal fibers.

they may take. It is interesting to follow these movements and the actions and reactions of the tissues in their natural physiological dependence upon each other and to recognize the forces at work.

When the teeth are malposed.

When teeth are in malpositions from some cause, and the proper devices are used to direct the teeth back into normal position — or better said, to stimulate the growth of the bones in such directions as to bring the features to the normal form and *allow the teeth* to come into proper positions, the walls of the alveoli about the teeth will *grow the changes* to accommodate the movement. In cases in which supernumerary teeth have diverted one or more teeth from their normal positions, they will come to their normal positions soon after the supernumerary teeth are removed; or if the cuspids have not fully erupted on account of lack of space, they will move into place if the proper space is made for them.

When we have learned the nature of these physiological relations of teeth, their alveoli and the bones which form them, and these forces are gently stimulated and directed, they do our bidding. This, taken as a whole, represents very briefly the physiological relations of the teeth and the growth of the bones of the face in which the alveoli and the alveolar process are active participants.

When teeth are extracted.

Finally, if further evidence were needed to show that the alveolar process is the physiological servant of its related tissues, and especially of the teeth, the results which occur when the teeth are lost may be cited. Straightway the alveoli are in part filled with a new growth of bone and the prominences of the alveolar walls are removed by absorption. Then a *residual alveolar ridge* is all that is left. In this there is no trace of the former alveoli. The gingivæ which rested upon the crest of the alveolar process, with all of their appendages, are gone. The conditions of the formation of this residual alveolar ridge, the influences which give good form and which give bad form, are very important. They are discussed elsewhere.

Results of a break in the peridental membrane.

Another point of importance that we should know early in our study of pathology is that there will remain no alveolar

process over any part of the root of a tooth without a peridental membrane. The peridental membrane makes the connection between the tooth and the alveolar process, and when this is broken in any part it is as if the tooth were lost, so far as that particular part of the alveolar process is concerned. Straightway this portion of the alveolar process is absorbed and removed.

Movement of teeth subsequent to extractions.

Another action which often does almost incalculable harm is apt to follow the extraction of any one of the teeth. Suppose, for instance, that a first molar is extracted when the person is twenty years old and the formation of the arches is practically completed. The socket of this tooth, which is the broadest in the mouth in the mesio-distal direction, is quickly filled in with bone, and its prominences, with the gingivæ which rested upon them, are removed by absorption. In the gum tissue which covers this, a hard, dense cicatrix is formed. The fibers of the peridental membrane, which formerly passed from tooth to tooth over the crests of the alveolar septal processes, and which have been torn across about midway between the two teeth both to the mesial and distal of the extracted one, are then attached to this cicatricial tissue. This shrinks very materially as the rule. This shrinkage, with the pull of the trans-septal group of fibers, tends to drag the second and third molars forward, causing them to lean over to the mesial, so that their occlusal surfaces do not meet the opposite teeth properly. The bicuspids may be similarly drawn distally. The result is bad occlusion of these teeth, which is liable to lead to their loss some time in the future by inducing disease, because of the derangement of contact points.

THE SALIVA

The saliva is a mixed fluid, the most important constituents of which are ptyalin, mucus, albumin and water in variable amounts, containing in solution the following salts: potassium and sodium chloride, potassium sulphate, sodium carbonate and calcium carbonate and phosphate. Certain others are frequent constituents, but not always present in appreciable quantities. Several of these constituents may be present, or absent; these may assume some importance in general descriptions. The presence of potassium sulphocyanide has given rise to a good deal of discussion in connection with the study of immunity to dental caries. The following data will give a better view of the constituents of saliva:*

Water		994.203
Solids:		
Mucin and epithelial cells	2.202	
Ptyalin and albumin	1.390	
Inorganic Salts	2.205	
	——	5.797
		1.000.000

(Potassium sulphocyanide 0.041.)

It might be said, then, that the saliva consists of water, in which there are suspended ptyalin, mucus, albumin and the salts which are mentioned above, and any one of them may be abundant or scant. The amount of these constituents is very variable.

I shall not in the present writing undertake any extended nor very critical description of the saliva, neither of the mixed fluids nor of its constituents, but shall give only an outline of these fluids as they are observed in the mouth in the practice of dentistry. The composition of the saliva is very complex, as will be seen from the table, but really it is very much more complex than the table would indicate, by reason of its great variability.

Some specimens of saliva are very mucilaginous, others are very thin and watery. Certain specimens seem to have large

* American Text-Book of Physiology. Wm. H. Howell.

amounts of albumin, others seem to be almost destitute of albumin. The albumin, mucus and other similar substances are known as the colloids of the saliva. In most specimens of saliva, scattering spherules may be found which have generally been spoken of as salivary corpuscles. In some specimens these are very abundant. These will be discussed a little later.

The loading of the saliva with carbon dioxid is practically continuous, and there are some other gases generally present in the fluid. Indeed, from the fact that carbon dioxid is produced in the blood and tissue juices in the metabolism going on in the body, and is excreted mostly by the lungs, it will be seen that this amount will be variable within certain limits, but that the gas will also be present in the secretions and excretions. The saliva always contains a variable proportion of this gas, which may be removed from it by reducing the atmospheric pressure on the liquid by the use of an air pump, and its quantity may be determined. The relative quantity of the other constituents of the saliva may be determined by chemical processes. For a full description of the saliva constituents, I would recommend the student to works on physiology, and especially the American Text-Book of Physiology, edited by Prof. Wm. H. Howell, Ph.D., M.D. This statement, with a general reading of one or two recent books upon physiology, in which studies of this subject are given *in extenso,* will prepare a student for special observations which I shall detail here.

Ptyalin.

Ptyalin is a digestive body found in the saliva. It is an unorganized, ferment body, or enzyme of the amylolytic type, which induces a peculiar action in starch, converting it into sugar. This action is produced very quickly in cooked starch, but in raw starch is so slow as not to be appreciable in the ordinary chewing of food. The effect of cooking upon starch is to break the membrane of the starch granule and expose the starch immediately to the action of ptyalin; while in raw starch the granule is surrounded by a membrane of cellulose, which prevents the ready action of the ptyalin upon the starch enclosed. Hence, much greater time is required for the digestion of raw starch. The action of the ptyalin of the saliva upon cooked starch is so prompt that it may be readily appreciated by taking a piece of ordinary bread in the mouth and chewing it. In the first acts of chewing this bread (taking no water), it will be

Fig. 112.

Fig. 112. A section cutting diagonally through the root. A, Network of epithelial cords; D, dentin; Cm, cementum. *Noyes.*

Fig. 113.

Fig. 114.

Fig. 115.

Fig. 113. Strings of epithelial cells from peridental membrane. From a section taken horizontal to the surface of the cementum, but a very slight distance from it. Cross cuts of these are seen at c, c, in Figure 109.

Fig. 114. Transverse section of the peridental membrane in the gingival portion, showing the position of the epithelial cords. The loop at A is shown more highly magnified in Fig. 116. *Noyes.*

Fig. 115. Epithelial cells from near the gingival border of the peridental membrane. a, a, a, Individual epithelial cells. b, b, Capillary vessel.

Fig. 116.

Fig. 116. Epithelial structures: Ec, Epithelial cord, apparently showing a lumen. Cb, Cementoblasts. Cm, Cementum. D, Dentin. This loop is seen in Fig. 114. *Noyes.*

FIG. 117.

FIG. 117. A famous dental cartoon, of Rowlandson, published in 1787, depicting the operation of transplanting teeth from the mouths of the poor to those of the wealthy. This operation was made popular by the publication in 1778 of the work of Sir John Hunter, entitled "A Practical Treatise on the Diseases of the Teeth," in which he describes the operation.

According to the historian Guerini, Abulcasis, an Arabian, 1050-1122, first mentioned replantation, but to Amboise Parè, 1517-1592, credit should be given for having first performed and described the operation of transplantation. Original of this illustration in Northwestern University Dental Museum.

FIG. 118.

FIG. 119.

FIG. 118. Reproduction of a radiograph of an upper central incisor implanted by Dr. Thomas L. Gilmer. When this radiograph was taken the tooth had been in the alveolus nearly three years. It will be noticed that the tooth had been eut away by absorption from either side almost to the root filling.

FIG. 119. A bicuspid tooth which was implanted and remained in the alveolus about three years. The extensive absorption of the root is very clearly shown. Specimen from Northwestern University Dental Museum.

Fig. 120.

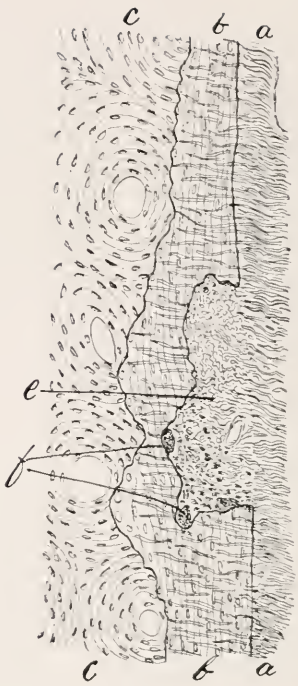

Fig. 121.

Fig. 120. Portion of the labial alveolar wall of an incisor that is being absorbed. a, a, Portion of the inner layer of the periosteum. b, b, Bone forming a portion of the labial wall of the alveolus. It will be observed that it contains a number of Haversian canals, h, h. e, e, A portion of the peridental membrane. d, d, d, Osteoclasts which are in the act of removing the bone, thus widening the alveolus. e, Space from which a large osteoclast has probably fallen during the preparation of the section. It will be noticed that where the osteoclasts are removing the bone, the fibers of the peridental membrane are detached and some little space is occupied by tissue of embryonal type, but in the spaces between the groups of osteoclasts the fibers are firmly attached to the bone. At f, there seems to be a little new bone formed to which fibers are attached. In this way bone seems to be removed, part by part, and the attachment of the membrane maintained.

Fig. 121. Portion of the alveolar wall of a cuspid tooth of an old person, showing absorptions. a, a, Portion of the peridental membrane. b, b, Portion of bone that seems to have been built on to supply an area of previous absorption. e, A recent absorption area. At f, three osteoclasts are seen. It will be noted that the fibers of the peridental membrane are detached throughout this area of absorption and the space is occupied by tissue of embryonic type. It should also be noted that the Haversian systems of the bone had been cut into by the previous absorption, removing portions of the rings of the Haversian systems. Residual fibers are seen in the bone b, but there are none in the Haversian bone c.

noted that it is moistened by the saliva. In a very short time, the chewing proceeding, a sweetish taste will be noted, and proceeding further with the chewing, until the bread is converted into a pulp, this sweet taste becomes a prominent feature. This is the result of the conversion of the starch into sugar by the ptyalin of the saliva, and is an experiment which any one may try and get an appreciation of the quick action of this body upon the starches. As quick as this action is, the reaction upon starch is never complete in the mouth, and when it passes into the stomach the action of the hydrochloric acid, which it meets with there, destroys the further action of the ptyalin and a final completion of the digestion of the starch is performed by the digestive bodies of the pancreas, especially the amylopsin, after its arrival in the pyloric portion of the intestine. It is said the ptyalin usually acts upon the starch alone, but I think this is not quite true. There is some action upon other ingredients of food as well, but it is so slow in the ordinary chewing of foods as not to be very appreciable and can not be reckoned with as a general action of this digestive body. This is the first act of digestion, which takes place when food is introduced into the mouth, and is practically the only act of digestion which occurs within the mouth itself.

The action of ptyalin in the mouth, however, is very much wider than that represented here in the chewing of starchy foods. The starchy foods are pasty and they are certain to stick more or less about the teeth and in the embrasures, and also in the interproximal spaces, if the septal gingivæ are a little bit short. After a meal the effect of the ptyalin in dissolving starch will clean up all of these pasty masses and remove them by solution in a very short time. This is another of the very important influences of ptyalin on the health of the membranes of the teeth. If it were not for this reaction of ptyalin, microorganisms would grow very luxuriantly in these pasty masses and would thus increase the injury to the tissues in all cases of irritation or inflammation.

Ptyalin is common to man and to the herbivorous and omnivorous animals, but is generally absent in the carniverous animals. From observations I have made the domestic dog seems to have developed a secretion containing some ptyalin. This perhaps has been developed by its habit of eating cooked starches with which more or less greasy or oily compounds are mixed. This probably does not occur in the wolf, the progenitor

of the domestic dog, and does not occur in the cat family, nor indeed in any other animal which is limited to flesh as its diet.

Mucus.

Mucus is one of the principal colloids of the saliva. It has a close resemblance to albumin and is probably a nucleo-albumin in its general make-up. It must be understood that mucus appears throughout the body and body juices, and its presence is seen mostly upon what we term the mucous membrane throughout the mouth and the digestive tract, also in other regions, such as the nasal passages, the trachea, the tubes of the lungs, the urinary bladder, and in other cavities of the body. In these various positions, there is much difference in the character of the mucus, depending upon certain elements in its combination. We will not go into these questions here, but will discuss only those which are of special interest to the dentist, and avoid all of the chemical questions, further than those included in this statement. Any one who wishes to pursue this subject further will find it fully exemplified in the more complete works on physiology. I would refer the student particularly to Howell's American Text-Book of Physiology, page 1019.

The saliva contains a considerable proportion of mucus. When this is abundant the saliva will be sticky and ropy. This ropiness may in a degree be determined by touching the finger to the saliva and drawing it away, noting how far a thread of it can be drawn. In very ropy saliva we may sometimes draw out this thread to the arm's length. In other saliva again, where the mucus is scant, one can not draw out a thread more than a very few inches. These differences will present the different proportions of mucus in the saliva.

The mucus is formed in the salivary glands. Only cells, or groups of cells, in the glands secrete the mucus; other parts of the glands secrete the watery portion. The mucus is immediately dissolved in the watery secretions from other portions of the same glands and flows through the ducts and is discharged into the mouth in this mixed form. There are other mucous follicles in the mucous membranes of the mouth, in some regions very plentifully distributed, which most of the physiologists whom I have read ignore. These are particularly plentiful in the fauces, and serve especially to lubricate the bolus of food for swallowing. There are also mucous glands scattered through the mucous membrane of the mouth, but these are less plentiful. From these one may see mucus in its pure form,

unmixed with the saliva. In the mouths of persons who have a considerable amount of mucus in the saliva, these mucous glands of the mouth are unusually active. If one will wash the roof of the mouth with a jet of water from the syringe, then dry it with a napkin, and place the finger in such a position that the tongue can not wipe off and rewet the roof of the mouth, one will note at least a few little globules of mucus appearing. If one of these is touched with the finger, and the finger is then slowly and carefully drawn away, one may be able to pull out a thread of this mucus a considerable distance, possibly as long as the arm will reach.

This represents the peculiar characters of the mucus. It is sticky, hangs to everything it touches, and when mixed with the general fluid of the mouth makes that ropiness of which some people complain. The mucus is the substance in the saliva which coats over the teeth, the mucous membrane, and every part of the mouth, and gives the slippery character which we may feel with the fingers during an examination. All of the mucus may be removed in a few moments by jets of water from the syringe, and if the mouth is dried, this slipperyness will have disappeared. This is done more or less every time one drinks water, or more especially by rinsing the mouth thoroughly with water. The difference in sensation in the membranes of the mouth when covered with mucus, and that after the mucus has been washed away, is quite noticeable. The effect of washing the mouth is to remove the mucus. Within a very few minutes the surface of the mouth will be recoated with mucus.

The function of mucus is a mechanical one. Many physicians speak of this as the only function of the mucus, and usually speak of it only as the lubricant of the bolus of food for swallowing, rendering it slippery for this particular purpose. This is entirely too meager a description of the function of the mucus. The whole of the mucous membrane of the mouth is made slippery by the presence of mucus upon it. The teeth and every part of the mucous membrane and the gingivæ are coated with mucus. In the act of chewing it is intimately mixed with the food and causes the food to slip easily over the surface of the teeth, gingivæ, and the gums, and prevents most foods from sticking to these parts by its interposition between them and the mucous membrane. In this way it is incorporated into each mouthful of food and facilitates the act of mastication. We could scarcely chew food without it. It causes food, when

crushed by the teeth, to run smoothly through the embrasures to either side of the arch. It also, in the act of chewing, permits the food to be easily thrown back upon the teeth by the muscles of the tongue and of the cheeks. Then by successive closures of the teeth, the food moves back and forth repeatedly in the act of chewing. This is just as important a function of the mucus as is the lubrication of the bolus for the act of swallowing. It is true that this is a mechanical function, but the ordinary amount of mucus found in the normal saliva is entirely sufficient to perform this lubrication efficiently. Some persons, when very much tired out and suffering for the want of water, will remember the difficulty of taking food under such conditions. Lacking this slipperyness caused by the mucus, food becomes unmanageable in the mouth.

Albumin.

Albumin is not usually reckoned as a normal constituent of the saliva, but it is frequently present. I know of no function that this albumin is destined to perform in the mouth, under normal conditions, and I suppose it is there by accident or some perversion of the secretive processes. In some mouths it is quite abundant and in others it can be detected only by very close chemical scrutiny, the amount is so small. The indications of the presence of albumin in the saliva have never been at all carefully studied, so far as I know. It seems to have been regarded as unimportant by physiologists and pathologists.

A certain amount of albumin is found in the urine, and in other secretions as well, including the saliva, and it is regarded as a state in which albumin is being lost from the system through the secretions generally, but through certain secretions more especially. Albumin urea, however, indicates a diseased state of importance and is always looked for very closely by physicians.

Salivary corpuscles, so called.

A number of those writing of the saliva speak of salivary corpuscles found in it, and there has been some speculation as to what these were and what function they might have. Most of our physiologists speak of them as the remains of leucoytes which have wandered into the saliva and are undergoing disintegration. I have searched a considerable number of the more recent, and some of the older, works on physiology, and find no mention in any of them of the existence of globulin in spherical

form. My search, however, has not been exhaustive, and I may have missed some announcement of that fact. They have not attracted very general attention, however.

In the course of my work I happened to run onto these, and by comparison it was easily made out that they were primary spherules of globulin, the same as the spherules that are poured out with the saliva in the formation of salivary calculus, which will be described later. I requested my laboratory assistants to determine the matter of identity or nonidentity by staining methods, in which they found that the two spherules were identical in their reaction with a considerable number of stains; and as they looked alike and were the same size, they were satisfied that they were actually the same. Therefore, I may state from my own determination made in a similar manner, and from the determinations as repeated by my assistants, that the so-called salivary corpuscle appears to be a spherule of globulin.

They are generally round, but not always a perfect sphere. They have no limiting membrane, and the margins are not infrequently somewhat ragged. They may be seen without staining, and are fairly translucent, the central parts showing dark, while the margins show light. The finding of many of these in the saliva indicates that globulin is being poured into the mouth with the saliva.

THE INVESTING TISSUES OF THE TEETH—GINGIVAE, PERIDENTAL MEMBRANE, CEMENTUM AND ALVEOLAR PROCESS

DISEASES AND TREATMENT

IN the consideration of disease beginning at the gingival margin of the peridental membrane the conditions might seem to be very different from those associated with beginnings of dental caries, death of the pulp and alveolar abscess, yet the care of the mouth necessary to the prevention of all of these conditions is practically the same.

We may find inflammatory conditions in the gingivæ of the child, which, while they need attention, may be regarded as somewhat trivial in that they tend to get well with little difficulty as a rule. Serious disease of the gingivæ does not very often occur in children. During childhood the free gingivæ are abundant and cover a considerable portion of the crowns of the teeth. The portion of the tissue in which inflammation begins is usually that which laps upon the crowns of the teeth, rather than the deeper portions lying nearer the gingival line. It is the extension of inflammation, involving the tissues at the gingival line that is most dangerous to the future of the teeth. While all of this is true, inflammations of the gingivæ of children should be guarded against as much as possible, and cases occurring should have prompt treatment.

Beginning with early adult life the greatest care should be taken as to diseases of the gingivæ, for at that time the gingivæ have shrunken to their normal length for adult life. Injuries are then apt to become more serious and suppurations are liable to begin at the gingival line, and cut away the tissues, forming pus pockets. After this has occurred and the pockets have made considerable progress, a cure becomes practically impossible. To be effective, whatever is done in the way of prophylaxis

against this disease must be undertaken before such an occurrence. This requires of the dentist that he make careful examination of the gingivæ of every patient, and if there are inflammations they should have immediate and most painstaking treatment. No matter what may be the cause of such inflammation, it should be searched out to the limit, found and removed. In this way I am persuaded from past experience that the vast majority of these cases can be prevented by removing the danger in its inception.

This requires quite as close a watch of patients as that necessary to prevent diseases of the pulp in children. There should be a wide range as to the frequency of the examinations of different patients. Some should be seen regularly every two or three months, others even more frequently, while others present a degree of health of these parts which will permit of the examinations being placed more widely apart.

The impression made upon the patient as to the importance of this condition has very great value, and should be carefully made by the dentist, so that the patient will not neglect to consult the dentist as often as may be desired for examinations. These little inflammations — and they often are seemingly trivial — are usually painless and they may pass on to a suppurative state, which will do great harm before the patient will realize that anything is wrong. The insidiousness of this class of diseases is such that not many, even dentists, have been regularly in the habit of noticing them during the early stages. A little redness here or there seems to be of no consequence, and after a time when the case has gone too far for remedies to be effective, the dentist will find the disease very serious and incorrectly suppose that it is comparatively recent in its beginning.

The matter of the deposits of salivary calculus about the necks of the teeth and impinging upon the free gingivæ, is also important. This will cause inflammation of the gingivæ and result in shortening and blunting of the margins or crests of the gingivæ, and even when they are brought to a healthy condition after removal of the calculus, they will not be so good as they were before. The very thin margins, which formerly came up about the teeth, will have become thickened so as to form better lodging places for débris and calculus. Each considerable deposit that is allowed to harden in such position gives its increment of injury to the tissue, making it worse and worse as to the collection of lodgments. For this reason every patient, in whose mouth a deposit of calculus is discovered, should be

trained to prevent this and avoid the injury which occurs as a result of occasionally permitting the calculus to become hard.

Finally, the whole question of prophylaxis as applied to this condition depends upon the practical care of the dentist, his training in the observation of the inflammations of the gingivæ caused by slight deposits, and his influence in bringing his patients to a realization of the danger, as an inducement to them to adopt certain systems of personal care calculated to prevent the occurrence or recurrence of such inflammations. This will be taken up step by step and developed along the lines indicated by modern research. Observation has led me to believe that the injuries caused by such deposits may be prevented in the mouths of practically all persons.

Brief Historical Review of the Development of Our Knowledge of the Diseases of the Investing Tissues.

As I look back over the field, the dental profession has never been disposed to give the diseases of the gingivæ and peridental membrane accurate and careful study as to pathological conditions. For what seems to me to have been a long time after I began practice, no attention was, within my knowledge, given to these conditions, except to remove calculus when patients appeared with incrustations upon their teeth.

Names applied to diseases of the investing tissues. The group of diseases of the investing tissues of the teeth have, without differentiation, had more names than any other group of pathological conditions occurring in the mouth, and new names are being continuously introduced. The earliest writers were content with the terms spongy gums, inflamed gums, loosening of the teeth, or others of similar import. Generally, the entire subject was disposed of in a single paragraph, or at most in a page or two.

In the sixties and early part of the seventies, Dr. J. M. Riggs, of Hartford, Connecticut, brought this subject prominently to the notice of the profession by clinics which he made before dental societies, rather than by writing. For a time Dr. Riggs' methods had quite a following, and the condition came to be called *Riggs' Disease*. This term is still seen occasionally in the literature.

The term *Pyorrhea Alveolaris* was proposed by Dr. F. H. Rehwinkel, of Chillicothe, Ohio, in a paper before the American Dental Association at its meeting in Chicago in 1877. It may be said that this name has become the most popular term for

the group of diseases of the tissues investing the teeth. The term pyorrhea alveolaris means the running of pus from the alveoli. It covers too much and does not properly describe the disease to which it is applied. I do not intend to use this term in this writing. This is not from any captious objection to it on my part, but because I wish to describe the several diseases under different names to distinguish them and make each as clear as possible. This I could not do under the single term pyorrhea alveolaris. In studies of the cast of mind of people, dentists as well as others, it will be observed that in order for a particular thing to be understood as a separate and distinct entity, it must have a distinct name, and this is as true of disease as it is of anything else.

In 1882, in a paper* before the Illinois State Dental Society, I proposed the term *Phagendenic Pericementitis* to apply to that form of disease in which pockets were formed alongside the roots of the teeth. This term goes no farther than to locate and describe the destructive character of the inflammation of the peridental membrane, a fact well known. This term phagedenic was formerly much used in describing certain ulcers which refused to heal and tended to the progressive destruction of the soft tissues. It means to devour or destroy by eating away.

The term *Chronic Suppurative Pericementitis,* which means the same and is more readily understood, will be used in this book.

In the paper referred to above, I proposed the terms *Calcic Gingivitis* and *Calcic Pericementitis,* as describing the form of disease caused by accumulations of salivary calculus upon the teeth. I believed the conditions presented well worthy of this distinction. Similar terms will be used in this book as a part of a simple classification of the several types of inflammation which will be described.

The term *Alveolitis* was, I believe, first used by Dr. Adolph Witzel, of Germany. It is being used by a number of American writers and is frequently seen in the literature. If this term, by its original definitions, had been made to apply to the soft tissues within the alveoli of the teeth — the peridental membrane — it would not have been far from correct. But it seems to have been proposed and defined with the idea that the principal seat of the disease is in the margins of the alveolar processes. This is, to my mind, an incorrect statement of the conditions,

* Phagedena Pericementi. Proceedings Illinois State Dental Society, 1882, p. 93.

as the alveolar process is the last of the investing tissues to become involved. *Dento-Alveolar Pyorrhea* and *Interstitial Gingivitis* are occasionally seen in the literature, as are other terms referring to diseases of the peridental membrane, which have their beginning at the gingival margins.

Dr. Riggs' treatment. One of the oldest discussions of Dr. Riggs' treatment occurs following a paper on Salivary Calculus read by Dr. Thos. B. Hitchcock before the Connecticut Valley Dental Society in 1869, and reported in the Dental Cosmos, Vol. XI, 1869, p. 412. At this time Dr. Riggs brought before this society a patient, Dr. Goodrich, for whom he had operated before this same society two years earlier. Dr. Riggs stated that he had been operating in the same way for twenty-five years, and the majority of his cases were successful.

In those days the essays and discussions of the Dental Societies were not reported so fully as in more recent times, and it is very difficult to trace the date of origin of important discoveries or events which occasionally become of special interest.

Dr. Riggs made some important statements regarding his treatments, which were published in the Dental Cosmos in 1882, p. 524. This was five years after the paper by Dr. Rehwinkel, and when the treatment was rapidly slipping away from the plans adopted by Dr. Riggs. His operation for the cure of these conditions was very simple, and without differentiation between the inflammations caused by deposits of salivary calculus and those in which pus pockets were formed. In practically all cases the operation was the same, except as to the extent of cutting required. The gingivæ and gum tissue were cut away sufficiently to remove all diseased tissue to the line of attachment of the peridental membrane. It was usually necessary to cut away more or less of the uninflamed gingivæ of neighboring teeth in order to have a reasonably even line of attachment afterward. This operation will often leave considerable of the cementum exposed when cases have healed.

It seems that many of Dr. Riggs' cases did well. In my own use of this plan in some very bad cases caused by deposits of salivary calculus, in which there was a great thickening of the gum tissue, I have been surprised at the rapidity of recovery and the readiness with which the soft tissues accepted the new line of attachment. The treatment was bloody, often extremely so, but the hemorrhage ceased promptly and was of no conse-

quence. Generally no medication was used. Whatever else Dr. Riggs may have accomplished, he certainly succeeded in calling the attention of the profession to the treatment of diseased gingivæ as had been done by no one else.

Dr. Rehwinkel's paper. Dr. Rehwinkel's paper in 1877 was by far the best writing upon this subject up to that time. It abounds in inquiry rather than in the discovery or announcement of principles of pathology. It is rich in references, and especially in quotations from both American and European authors. Taken altogether, it gives a good view of the opinions of men regarding the diseases of the gingivæ at and before the time at which it was written. Although it set a landmark in the name to which it gave origin, it did little to advance our knowledge of these diseases. A feature of the paper that seems curious to one reading it now is the fact that no hint is given of the pus pocket as such, or as a distinct entity in the pathology of the peridental membrane. The nearest approach to this idea is the mention of the peculiar form of calculus which occurs on the sides of roots when the membranes have been destroyed. He states that this calculus is something different from salivary calculus. In his quotations there are many suppositions regarding systemic conditions as causative factors in inducing disease of this tissue.

Gouty diathesis as a theory. These events occurred at a time when there was the wildest use of antiseptics in the treatment of suppurative conditions wherever found, and often without much regard to other conditions. This may have caused the neglect of the study of the pathology which is so general in the writings on the subject. It is true that from time to time suggestions as to the pathology have been advanced. Many of these have been based upon suppositions regarding systemic causative influences, as a connection between the gouty diathesis or uric acid dyscrasia and pus pockets on the roots of the teeth. Perhaps an article* by Dr. Edwin T. Darby, of Philadelphia, in 1892, gives the best expression of this thought, which had a wide influence for a considerable time. The treatment for the uric acid dyscrasia seems to have been tried out very thoroughly by a number of practitioners. However, we may judge that such treatment has not been sufficiently successful to justify its continuance. Gout and rheumatism are eminently nonsuppurative diseases, while diseases of the peridental membrane are as

* Dental Erosion and the Gouty Diathesis. Dental Cosmos, Vol. 36, 1892, p. 629.

remarkable for their suppurative features. They stand wide apart.

In the course of the discussions referred to it was announced by Dr. C. N. Pierce* that he had found calculus on the sides of the roots of teeth not before diseased, about which abscesses occurred, and that these calculi showed uric acid by the murexid test. Very soon there were other cases reported. It then looked as if we should have to use this test upon any calculus found in the mouth as a diagnostic feature.

Under these conditions I undertook a bit of work in this line. I was skeptical regarding the finding of calculi on the sides of the roots of teeth as the initial step in the formation of pus pockets. The conditions causing lateral abscesses had been so persistently overlooked by others that I felt free to question this statement. The peridental membranes are often deeply diseased with but little showing superficially. One who is not habitually examining the subgingival spaces may readily overlook the existence of pus pockets until he is surprised by a lateral abscess.

I was at the time seeing many patients in various conditions of physical health; good, medium and bad. Some had diseased gingivæ, some had not. Some had rheumatic or gouty tendencies, some had not. From these I gathered calculus with written records, made the tests, and made a report† in 1894 as a reply to the findings of Dr. Pierce and others. I found the test entirely unreliable as showing a uric acid dyscrasia. It was occasionally absent when it should have been present, and was often present when it should have been absent. The fact seemed to be that the small amount of uric acid present in the blood in normal conditions might give color in salivary calculus by this test. It was therefore useless as a diagnostic feature.

Special infection theory. There has been for many years an almost incessant search for some special infecting agent which serves as an initial cause of the formation of pus pockets, and the gradual spreading of these through the mouth. This search, in which I myself was active for a long time, has not, up to the present time, brought definite results. No complete studies of the micro-organisms normal to the human mouth have yet been well presented in any book or writing. Dr.

* Etiology of Pyorrhea Alveolaris. International Dental Journal, Vol. 15, 1894, p. 1.

† "Diseases of the Peridental Membranes and the Uric Acid Diathesis." Dental Review, Vol. 8, 1894, p. 449.

Miller and others have isolated many species of organisms from the saliva, but we should know accurately what micro-organisms are normal to the human saliva, or may always be found there. All others found there will be in the saliva by accident, i. e., accidentals. These may or may not be pathogenic varieties.

Some years ago I took up the subject in this way. A certain number of plants were made from each mouth and plated out. Pure cultures of each organism, which would grow and form colonies on semisolid media, were obtained and their pathogenic properties tested by inoculation of animals. The distinct forms were listed, and divided into two groups; those which were normal or constant in the saliva and those which were accidentals. In this way I studied many mouths, trying to find the organisms normal to the saliva, or those present in every mouth which was well kept, and the additional organisms found in a series of mouths not well kept. These investigations showed certain organisms constantly present in the mouths of careless persons which could not be found in the mouths of persons who were careful as to cleanliness. Many other facts will appear in such a course of study which will surprise most bacteriologists, even those who believe themselves well acquainted with the flora of the human saliva.

About fifteen varieties will probably cover the organisms that are constant, only about half of which can be cultivated upon the ordinary media, semisolid or fluid. All of the others are accidentals. In one locality some of the accidentals may persist for one or two years, and then disappear. I found one organism constant in Chicago among both students and infirmary patients, for two years — 1892 and 1893. I had never seen it before. The third year it had disappeared completely. It did not occur in my cultures in Jacksonville, Ill., two hundred and forty miles away. Taking just this small line of facts, one will see that it is not safe to rush into print on such a proposition. To try out these organisms on animals as to possible pathogenic qualities, is in itself a large undertaking.

I found that, with my practice, I could not have the time to make this line of work complete. It is expensive and exacting as to both space and care to keep the necessary animals for pathological tests in such a way that they will not contaminate each other. We need young men with sufficient financial support to enable them to do this work. It is only by such work that we will ever obtain that broad view of this subject which is so

desirable. At present we are unable to definitely place any organism, except the one constant in the saliva, the staphylococcus albus, in a causative relation to the suppurative features of diseases of the gingivæ. When the inflammations are begun by traumatisms of any kind, this organism will keep up pus formation as long as there is a pocket in which it can remain enclosed. It is not a virulently pathogenic organism. It is common to the skin and is the organism generally found in boils. Without some break that gives it an advantage, it will not initiate a condition of disease in the mouth or in the skin.

Serum treatment. Serum for the control of suppurations is being sought for by many bacteriologists and other researchers. As a general principle it would seem that any infection which is self-limiting should be controllable by an immunizing serum. This is in accord with the theory that the poison eliminated by the causative organism arouses the formation of an antibody by the tissues themselves, which destroys the effect of the micro-organism. Such a serum may be employed to establish immunity, or it may be administered soon after exposure or at the beginning of an attack, and either prevent the attack entirely or materially moderate it. In this group of diseases a more or less permanent immunity is established by the use of serum.

In the use of serums to control suppurations, it is not expected that immunity will be established. The patient may be cured of the particular attack, but there will be no lasting effect against another similar attack. Many cases of peridental disease have been reported as much improved by the use of serums, the same as pyogenic infections in other parts have been benefited. There has, however, been no cure of the pus pocket by such treatment, even though the discharge of pus might have been temporarily stopped. The denuded cementum has remained as a continuously acting irritant, and as soon as the effect of the serum has passed, conditions are favorable for the re-establishment of the infection and pus formation.

Regardless of the success of this and other methods of treating suppurative infections up to the present time, or of the further progress of this work in the future, it does not appear to-day as though we can hope for relief in the application of these methods to the pus pocket alongside of a root, for the reason mentioned above, that we are unable to remove the continuous irritant.

The treatment in vogue. All of this has had but little influence in shaping the general treatment now in use for chronic suppurating pockets. The treatment generally in vogue has consisted of the removal of deposits of serumal calculus which were found adhering to the cementum where the soft tissues had been parted from it, and the use of antiseptics in an effort to control the discharge of pus. The supposition was that the soft tissues would become reattached to the cementum if they had a favorable opportunity. This, it was thought, would bring about a cure of the condition. This is a succinct statement of the practice now most generally employed in pus-pocket conditions.

Has this treatment proved satisfactory? Have cases presenting deep pus pockets on the sides of the roots of teeth healed and remained well afterward under reasonable care by the patient? In this, it is not a question whether cases come to look better and to show less flow of pus under this treatment, but do they really get well with a reasonably good reattachment of the soft tissues to the cementum?

I have had a long and very careful observation of this treatment, both in my own practice and in the examination of patients who have been under treatment by others. I have found much improvement in general conditions of the gingivæ as examined by the eye. I have seen cases which had been bad, with much pus issuing, improve so that no pus was apparent. Such have been common in my own practice, and I am sure this is true in the practice of others also. However, a careful examination with the subgingival explorer showed that the pockets had not closed, and subsequent observation revealed the fact that the tissues of the gingivæ had not maintained a healthful tone.

I have myself kept patients on and on in this condition, all of the time having them make frequent visits for inspection and direction as to cleaning, all of the time making frequent use of antiseptics. These cases have apparently done fairly well in the main, but have never really gotten well by reattachment of the tissues to the cementum. Some of them would show only occasionally an acute inflammation about some particular tooth, or teeth, which passed away, leaving the pocket deeper than before. In this slow way the cases became worse.

In some of the cases I placed the loose teeth in bands connected with other teeth to hold the looser ones steady. As the roots became more and more difficult to keep clean, on account of the broadening of the already wide pockets, I cut off and

removed the roots, leaving the crowns in the gold bands to serve the purpose of mastication. This was certainly pushing the preservation of the natural teeth to the limit, and I came to so regard it. I have since had reason to believe that I went much too far in my effort to cure. Much too large a percentage of those people are dead. As it was with my patients, I believe it has been with patients of others. After the use of the forceps I have seen many of these sallow, not much sick but complaining persons, brighten up and again enjoy life.

STUDIES OF SALIVARY CALCULUS

ILLUSTRATIONS: FIGURES 122-143.

Salivary calculus is the term applied to the calculus which enters the mouth with the saliva, and becomes deposited upon the teeth, plates, or other hard substances within the mouth. The word salivary is used to distinguish this deposit from the calculus which may be deposited in the gall bladder, urinary bladder and elsewhere. Since other calculi found in the body are very closely related, the gall bladder calculi being formed of cholestrum instead of calcium salts, and since the underlying causes of all are probably similar, the investigations here presented apply in large measure to all of the various forms of calculi. In fact, it will be shown that there is little question to doubt but that the elements necessary to the formation of the deposit in all the various places in which it may occur are present in all simultaneously and lack only the local nidus which is necessary to a beginning accumulation.

Composition.

Salivary calculus is composed of calcium phosphate, with the addition of smaller amounts of calcium carbonate, held together in mass by an organic compound which, according to general opinion, is formed after the material has been deposited upon the teeth, natural or artificial. The fresh deposit is very soft and greasy to the feel of the fingers, insoluble in water, in alcohol and most fluids that one would be likely to try. This mass I have called *agglutinin,* or *agglutinin of calculus.* When it is deposited upon a plate, or upon the teeth, it may readily be washed away and the plate or the teeth perfectly cleaned with an ordinary brush and water. This, however, must be done within five to twelve hours after the material has been deposited, if it is to be removed easily. If one waits twenty-four hours it has begun to harden and it is difficult to remove with the brush. If one waits for several days or a week, it can not be brushed away. It continues to increase in hardness for one or two months, and at the end of this time is fully hard. When it has become hard, instruments are required to break or to scrape it away.

The deposit does not occur in every mouth. In some mouths, there will be an occasional deposit, with long periods during which there is none. In others the deposit seems to be occurring all the time. Generally children and young people are freer from deposits of calculus than adults. Quite a number of cases occur in which persons have no deposits until they are forty, fifty or even sixty years old, and afterward are much troubled with it. These may be said to be general conditions noted by every dentist who is a good observer and has had many years of practice. It gives the idea that there is a systemic dyscrasia which is responsible for these deposits.

Analysis. In the text-books, a number of analyses of salivary calculus have been published. These differ considerably. A part of these differences are due to variations in the amount of water and mucus, and the fact that a number of them combine water and organic substances in their report. One may analyze calculus fresh from the mouth, only drying it upon blotting paper. Another may have analyzed calculus that was old and thoroughly dry. Unless these conditions are stated, the amount of water will vitiate the figures of the whole analysis. I give here an analysis by Schehevetskey* which gives as good an idea of its composition as can be obtained from these analyses. Such reports would naturally vary, for I do not suppose calculus is a strict chemical compound of invariable composition.

Water and organic matter	22.07
Magnesium Phosphate	1.07
Calcium Phosphate	67.18
Calcium Carbonate	8.13
Calcium Fluoride	1.55
	100.00

In a number of the analyses no magnesium phosphate is reported; in some a little calcium fluoride is reported.

STUDIES OF DEPOSIT OF SALIVARY CALCULUS.

Considering the length of time in which the deposit of salivary calculus and the great injury it has done to mankind have been observed, the history of the study of it in the literature

* Burchard on "The Origin of Salivary Calculus," Dental Cosmos, 1895, p. 821. Also see Burchard's Dental Pathology, edition of 1898. A considerable number of these analyses are very old, and may be traced from book to book from away back to the first half of the last century, or even earlier. I have noted in a few of them that errors in transcribing have occurred, and have been carried on from one book to another.

is very disappointing. Until quite recently it would seem that no successful effort has been made to penetrate this mystery. All, or nearly all, have agreed upon certain points and there the subject has been dropped. The essential facts in the writings of many men may be covered in a few sentences.

The points on which most men have agreed have been these: Calculus is composed mostly of calcium salts which are precipitated from the saliva. These salts find lodgment and settle in out-of-the-way places about the teeth, and become aggregated by entanglement in partly inspissated mucus or other colloids from the saliva. In these positions the material settles into more compact form, as the colloid material is slowly decomposed, and hardens into stone-like masses. These masses grow by more or less constant additions upon the hardened or hardening material, until, sometimes, quite large and thick masses of it are formed. These masses are in part in contact with the soft tissue investments of the teeth, and cause them to become inflamed and to be destroyed partly by absorption and partly by suppuration, resulting in the loosening and final loss of the teeth.

Among the writers much difference of phraseology may be observed, but the whole subject is practically included in the above statement. The late Dr. A. W. Harlan was to prepare a paper on salivary calculus for the American System of Dentistry which was published in 1886. In a conversation regarding this paper he stated that there was little to write, as the subject really had no literature, and nothing was positively known about it. He finally offered an article which is printed in the second volume, page 273 of that work, consisting of nineteen pages. Four of these are taken up in quotations from thirteen different authors, two to a discussion of green stains on teeth and the remaining pages to the removal of calculus from the teeth. This statement strikes me as the most graphic representation of the *little that was known* of the subject up to that time, that I could now write.

Dr. Burchard's studies. Since the publication of the American System of Dentistry, further efforts have been made to advance our knowledge of this subject. The most notable of these will be found in Dr. Henry H. Burchard's article* on "The Origin of Salivary Calculus" in 1895, and in his book† published in 1898. In a second edition of the book, since Dr. Burchard's

* Dental Cosmos, Vol. 37, 1895, p. 821.

† Dental Pathology and Therapeutics, 1898, p. 447.

death, his ideas have not been very closely followed. From the frequent references to assistance and support by Dr. E. C. Kirk, of Philadelphia, it would seem that he had Dr. Kirk's assistance in the development of his subject, especially in the preparation for, and the carrying forward of, the experimental work, the results of which form the basis of the presentation in both the journal article and in the book.

In order to fully comprehend a short résumé of this work and its conclusions, one should have in mind the following well-known facts: Carbon dioxid dissolves in water or fluids containing water. When a fluid contains this gas in solution, its power of dissolving certain salts is markedly increased. The excess of salts thus dissolved above saturation without the carbon dioxid, will be precipitated if the carbon dioxid is removed. If the pressure of the atmosphere is increased — as by pumping carbon dioxid into a closed space with an air pump — the amount of carbon dioxid dissolved in the liquid is increased and its power of holding salts in solution is increased in a similar proportion. If now the pressure which holds the carbon dioxid in solution is relieved and the extra portion of carbon dioxid allowed to escape, the extra proportion of a salt dissolved under pressure will be precipitated.

In the animal body carbon dioxid is continually being formed by tissue metabolism and eliminated, mostly by the lungs, but always leaving a residue in the tissues and body juices. Therefore, all of the fluids of the body are, in a degree, charged with carbon dioxid, and its power of holding salts in solution is influenced to some extent by the blood pressure. Therefore, these fluids, as well as the secretions and excretions derived from them, may have a little more carbon dioxid and a little more calcium salts in solution than they will retain after removal of this blood pressure, which they lose when secretions which contain them are exposed to the air. In such a case the extra amount of calcium salts in solution, if there be any in excess of ordinary saturation, will be precipitated. It is only by experimental results that we can know whether such an excess exists in any particular secretion.

The propositions made by Dr. Burchard are reducible to two, around which the whole experimentation and argument hinges. These are:

1. Saliva contains calcium phosphate and other salts in solution, held by dissolved carbon dioxid. When delivered into

the mouth, the normal pressure on the body juices is relieved, the carbon dioxid escapes and a portion of the calcium phosphate and the other salts is precipitated.

2. Mucus is a normal constituent of saliva. Lactic acid is being continuously formed in the mouth by certain micro-organisms. Lactic acid converts mucus into a curd in which the precipitate of calcium and other salts becomes entangled, and this hardens in the form of salivary calculus.

These statements agree with the views generally held as to the nature of these deposits.

In the experimental work reported, it was found that freshly collected saliva cleared by filtering,* or otherwise, and placed in an open test-tube, will have become cloudy the next day. The interpretation was that the saliva, known to contain calcium salts in solution, slowly lost its carbon dioxid and the excess of calcium salts was precipitated and formed the cloud.

Another strong feature of Dr. Burchard's experimentation I may fairly express in this way. The mixed saliva contains a considerable proportion of mucus as a normal constituent. This mucus is precipitated by lactic acid in the form of a curd, which rises to the surface, the amount and strength of which will be in close relation to the strength of lactic acid used. This curd may be seen when a few drops of one per cent lactic acid are dropped into a test-tube of freshly drawn and cleared saliva. A much stronger curd is formed by a few drops of ten per cent lactic acid. The saliva of different persons and of the same person at different times contains a variable amount of mucus, and the amount of this curd will also depend upon the amount of mucus in a particular specimen of saliva, as well as the percentage of lactic acid added. The supposition expressed is, that the precipitate of calcium salts, falling out of solution, becomes entangled in the slight curds of mucus forming in undisturbed places about the mouth. These become harder and stronger, forming salivary calculus, which grows very slowly but in time forms solid, stone-like masses. These are in positions in which they can slowly settle without too much disturbance from the movements of the tongue and the buccal and labial mucous membranes, as along the crests of the free gingivæ, especially in places where there is a slight thickening of the crest, which will afford a little protection against the rubbing of the tissues and

* In the filtering of saliva much of the colloids remain on the filter, changing the composition in that degree. This is avoided by clearing with the centrifuge, or by allowing the saliva to stand until it settles.

of the food which is eaten. This idea is in some difficulty to account for the almost universal observation that the first and greatest deposit of calculus occurs on the teeth nearest to the ducts of the salivary glands.

The papers mentioned are well written and their appearance marked a new era in the study of this subject. Since their publication they seem to have served as the basis of thought for other writers.

I will show in the next few pages, however, that the cloud formed in the test-tube, supposedly calcium salts precipitated with the release of carbon dioxid, was not calcium salts but a growth of micro-organisms, and that salivary calculus is deposited in a substratum of globulin and not in a coagulated mucus; that a very considerable deposit may occur near the salivary ducts within a few hours instead of this slow settling down in the out-of-the-way places, etc. Again, lactic acid, which is added to saliva in a ten per cent solution in order to form a strong curd — as expressed in Dr. Burchard's account of his experimentation — would convert a precipitate of calcium phosphate into a highly soluble lactate, which would be carried away in the oral fluids instead of forming a hard concretion.

Again, the formation of what I originally called the gelatinuous plaque, gelatinoid plaque, etc. (always avoiding a strictly chemical term, of which I was uncertain), has apparently been found to be by the coagulation of mucin into a film or mass by lactic acid.* This is developed by the growth of a colony of micro-organisms occupying a sheltered position, and which is more perfectly covered in by this film. This condition prevents the acid formed by the organisms from being dissipated in the general saliva. The result is a solution of the calcium salts of the enamel, forming caries of the enamel. Hence, we see that the scene of the coagulation of mucin by lactic acid, is that of a solution and conversion of the less soluble phosphates into the more soluble lactates. This was well shown by Dr. W. D. Miller in his original experiments, by which he determined the phenomena of caries of dentin (American System of Dentistry, Vol. 1, p. 791). This again shows definitely that a coagulum of mucin by lactic acid would not become the scene of the entanglement of precipitated particles of calcium salts for the formation of the hard concretions, such as salivary calculus.

* "Some Conclusions Growing Out of a Study of the Cause of Dental Caries," by Charles E. Jones, Dental Review, 1911, p. 1167.

Therefore, it seemed necessary that we find some other explanation of the formation of salivary calculus.

Personal Investigations of the Deposit of Salivary Calculus.

The following report of my investigations of the deposit of salivary calculus is written after about five years of experimental work. Naturally many experiments were pursued with results which were of little or no value, others failed from one cause or another. For this writing I have selected and reported only those which have added something to our knowledge of the subject. These are presented in what seems now to be the most logical order, without regard for the order in which they were actually made.

During these investigations I have written two articles in which the knowledge of this subject developed at the time of writing was presented. The first of these, entitled "Beginnings of Pyorrhea Alveolaris — Treatment for Prevention, etc.," was published in the Items of Interest, Vol. 33, 1911, p. 420. The second, entitled "Deposit of Salivary Calculus," was published in the Dental Review, Vol. 26, 1912, p. 337.

A special machine was designed and built for the purpose of grinding microscopic specimens of hard substances, such as deposits of calculus, teeth, etc. This machine is described at the end of the book, and is illustrated in Figures 508 to 518.

As a basis for my experimental work, I duplicated the work reported by Dr. Burchard. I made the same experiments in filtering freshly collected saliva and observed the cloud which was present in the test-tube the next day — the cloud which had been interpreted to be composed of calcium salts precipitated by the gradual loss of carbon dioxid from the solution. It occurred to me that this experiment was unfinished; it had not been proven that the precipitate was calcium salts.

Test of saliva for precipitate of calcium salts.

I devised an instrument by bending a small wire in a loop and then twisting one of the ends around the other for a sufficient length to reach nearly to the bottom of the test-tube. The ends of the wire near the bottom of the tube were converted into a spring clutch, into which I placed a cover-glass which would nearly fill the inner circumference of the tube, in a horizontal position. This was let down through the clouded portion to within a half-inch of the sediment which had collected in clearing the saliva, without disturbing it. I then placed this test-tube in

the electric centrifuge, and allowed the machine to run fifteen minutes. On examination through the test-tube I found the liquid above the cover-glass clear and a film on the cover-glass. The fluid was now drawn away as far as the cover-glass with a pipette, so cautiously as not to disturb the film. Then the cover-glass was lifted out. When this was brought under the lens of the microscope, it did not show a precipitate of calcium salts. *The cover-glass had on it a film of micro-organisms.* This result occurred in every effort to prove the findings related. I have not yet been able to find the least trace of precipitated calcium salts, though I have made many efforts. I had previously fully believed that such a precipitate did occur and the results obtained were disappointing.

Examination of deposits on artificial denture.

My recent studies of the deposit of salivary calculus were undertaken soon after I began wearing a full upper plate, a little more than five years ago. My first plate presented the opportunity to observe the deposit of salivary calculus which I occasionally found upon it. After a time, I found some features which had never before been presented. I then began a systematized study of the conditions under which deposits occurred. At first this consisted of a record of the appearance of calculus in soft form upon the plate. In pursuing this, I soon discovered that the mucous coating which covered the plate and made it slippery to the fingers, could be washed away by placing the plate in still water for a time, or in running water, as a jet from the ordinary hydrant, without disturbing the freshest and softest deposit of calculus. The following then became the mode of examination: After each meal the plate was laid under the hydrant and the water turned on it for a few minutes, removing the mucus. It was then examined for deposits of calculus. These were found only occasionally. Sometimes two to four weeks, or a longer time, would pass without any deposit whatever. Then suddenly a heavy deposit occurred. After the washing with running water, and the examination, the plate was always made clean in every part. The deposit seemed absolutely insoluble in running water, hot or cold, yet it was so soft that it was readily cleaned away with a brush and water. Nothing more was needed.

Generally the deposit was divisible into three zones: a central greyish-white zone, an intermediate semitransparent zone and an outer transparent zone. The whole deposit felt greasy

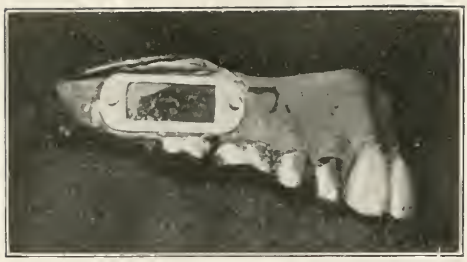

Fig. 122.

Fig. 123.

Fig. 122. A device attached to a plate for artificial teeth, used for the collection of specimens deposited directly on a piece of the usual cover-glass for microscopic objects. It consists of a frame of No. 26 gold plate fastened to the plate with a gold screw at each end. All of the central part is cut away, as shown. The vulcanite has been cut flat over the area covered by the frame, and above and below ledges are left which will prevent a glass cut to fit the space slipping out in those directions. The screws keep it from slipping out endwise. The cover-glass is laid in the space, the frame is laid upon it, and screwed down. This exposes all of the central part of the cover-glass for the collection of films. When a film has been deposited on the glass while being worn in the mouth, the screws are removed, the frame lifted off, and the cover-glass, with the film undisturbed, is removed and transferred to the liquids prepared for the staining process. A similar device may be attached to the natural teeth in such a way as to be removable.

Fig. 123. Photomicrograph of agglutinin of salivary calculus moderately well filled with calcium salts, but very soft. It was pressed down under a cover-glass in a thick solution of shellac in alcohol, after thirty minutes in alcohol to remove water. The general appearance of spherules is fairly well seen.

Figures 122 to 134 and Figures 138 and 139 were originally published in the Items of Interest, illustrating a paper entitled: "Beginnings of Pyorrhea Alveolaris, Treatment for Prevention, etc.," Vol. 33, 1911, p. 420.

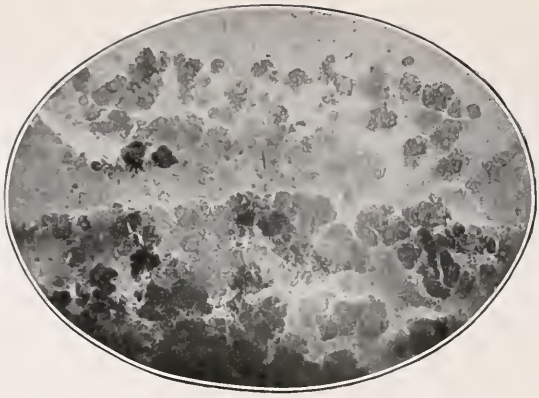

FIG. 124.

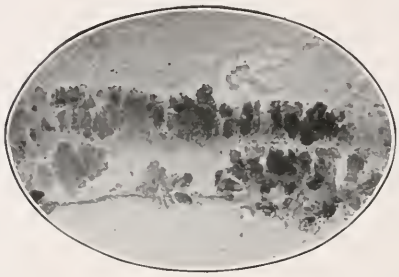

FIG. 125.

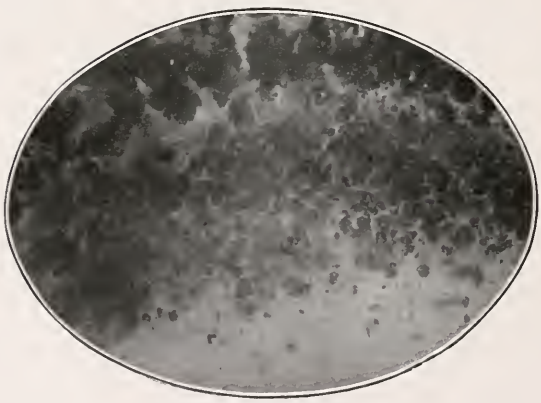

FIG. 126.

FIGS. 124, 125, 126. Agglutinin of salivary calculus showing irregular spherules laid down on cover-glasses worn in the mouth. Eosin stain with formalin as a mordant. The stain is diffuse.

In this deposit the thickest spherules show darkest. The finer spherules, of which these are made up, are not distinguishable in the pictures. The tendency to form larger spherules by the combination of smaller ones is apparent, but presents the utmost irregularity in the different specimens.

NOTE.— The beginning of the deposit always occurs in the little angle formed by the frame and the cover-glass (see Figure 122), and grows out upon the glass from that beginning. The cover glass has generally been removed before being completely covered, in order to have thin margins. All the specimens are so placed that that portion next to the frame is down in the illustration.

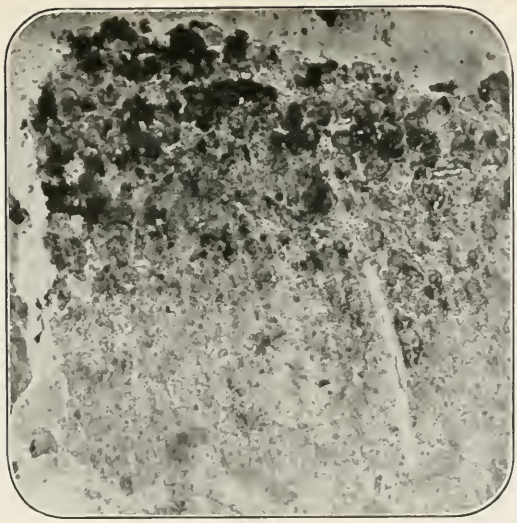

Fig. 127.

Fig. 128

Fig. 127. Ground section of hard salivary calculus ½ of 1/1000 inch thick, showing spherules in the upper part of the field. While these spherule forms are hard calculus, that portion has not received as much calcium salts as it would have contained later. The spherules finally become almost completely obscured, as seen in the lower portion of the illustration.

Fig. 128. Photomicrograph from a section of a crumb of very black serumal calculus. (See description of this process of grinding in the Appendix.) The outer surface is the lower border of the picture, upon which accretion was in progress. It gives a slight showing of spherules. The irregular veining shows lines of accretion.

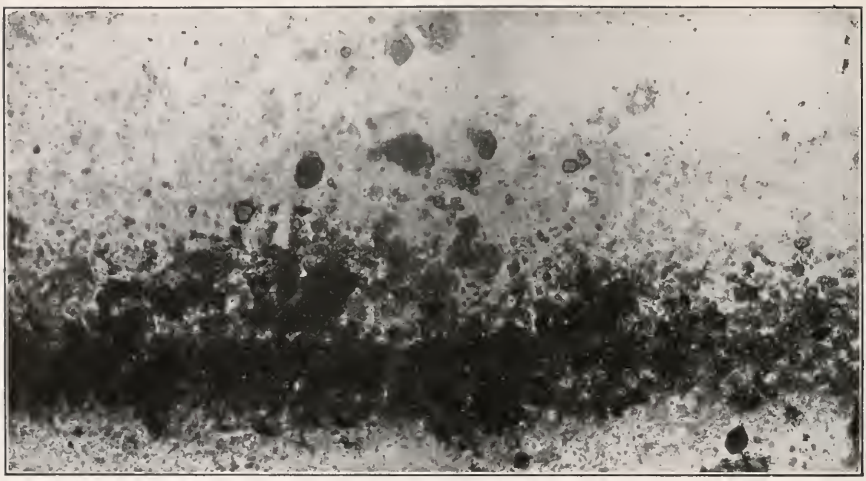

Fig. 129.

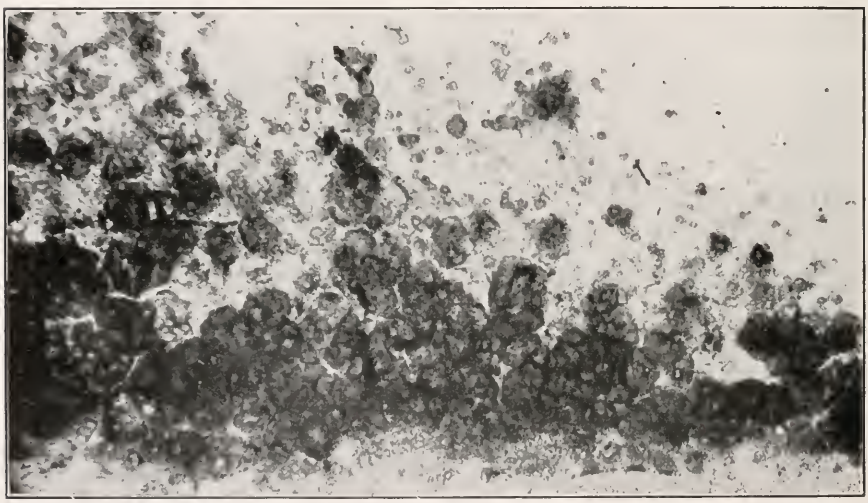

Fig. 130.

Fig. 129. A stain by nigrosin following phenol. Certain of the spherules do not stain at all; otherwise the stain is diffusive. A number of light-colored circles will be seen in the upper part of the field, which are unstained spherules, with a collection of fine granules about them that take the stain poorly. Many of these white spherules appear in the thicker portions partially covered with spherules that stain. Therefore their outlines appear irregular. Many of these peep through the thicker portions as white points.

Fig. 130. Appearance of a rapid deposit (about four hours), stained by nigrosin after treatment by formalin as a mordant. So far as the stain goes it is diffusive, but many of the primary spherules refuse the stain, which gives a lobulated appearance.

and sticky to the fingers. It was coagulated and whitened by boiling water in a similar way as is white of an egg — egg albumen. Phenol or alcohol produced a similar effect. Therefore, if this material is not albumen, it is something closely approaching it in chemical composition and reaction to coagulating agents. It seems clear that it is not coagulated mucin, or the settling of a precipitate from the saliva, like that in the teakettle in which hard water is boiled, as taught by Burchard.

Collection of deposit on cover-glass.

While my series of observations on conditions of deposit were continued, I made every effort to find means of displaying the structure to better advantage. Finally the idea that I might construct a trap by which I could catch the mass on a cover-glass, suggested itself, and was quickly carried out.

This trap consists of a little frame of gold plate fastened at either end with a screw, under which a cover-glass may be laid. (See Figure 122.) A space for it on the plate is cut flat, leaving square shoulders at either side to prevent the cover-glass from slipping out. The trap is depressed a little below the general level of the surface of the plate, in order that deposits on the glass will be less likely to be disturbed. The traps which I have used take in a cover-glass five-eighths by five-sixteenths of an inch. They may, however, be made of any size or form. At first cover-glasses were altered by grinding them on the emery-stone to fit the space, but later a dealer cut the special form for me.

A trap may be securely attached to one or two natural teeth in easily removable form. It has one advantage over the trap attached to a plate, in that it may be removed and dropped into water while eating, avoiding all danger of disturbing the form of a deposit in chewing food. One who wears a plate may have one plate to wear at meal-time and another carrying the trap to wear at other times. In this way the danger of disturbing the deposit in chewing food will be obviated.

On these cover-glasses I caught the fresh deposits in the form in which they were laid down, and soon learned to avoid those which were too thick for microscopic study. The deposit would invariably begin in the little angle formed by the meeting of the gold plate and the cover-glass, and spread from that out over the cover-glass. It was desirable to remove the cover-glass before it was completely covered with deposit, in order that a thin margin would be presented for study. The screws were removed, the frame lifted, and the cover-glass with the deposit

transferred to the fluids for washing, staining and otherwise preparing for mounting. When the material was mounted in balsam, without other preparation than the removal of water with alcohol, it became so transparent that nothing could be seen, except where it was thickly filled with calcium salts, and even there no form elements could be discovered. Therefore, some kind of stain became necessary.

Staining. I found that this material could not be stained by the ordinary processes for staining tissues or micro-organisms, for the reason that all of the stain would wash out. By using stains soluble in absolute alcohol, increasing the strength of the solutions, and leaving the specimens in them from twelve to twenty-four hours, then giving an hour or two in absolute alcohol for removal of excess of stain, fairly good selective and diffusive stains have been produced. A ten per cent solution of gentian violet in absolute alcohol, and a saturated solution of eosin in absolute alcohol, gave very satisfactory results. Gentian violet is a selective stain. Eosin is a diffusive stain. Nigrosin answers certain purposes very well, since it shows selections which other stains do not. Many other stains have been tried but have not given better results. By using formalin, four per cent solution first, as a mordant, the time required for staining is much reduced. Phenol, twenty per cent, produces a similar result.

As to the use of the stains mentioned, eosin is good for showing the *general forms* of the masses composed of spherules. (See Figures 124, 125 and 126.) The gentian violet is a selective stain and shows the *structure* of the larger masses of spherules. (See Figures 131, 132 and 133.) This structure is made up of several kinds of spherules, i. e., spherules differing chemically, and because of these individual differences, take and hold the stain differently and show the individuality of certain similar spherules by similar stains or differentiations by different degrees of color. Some of these stain very brightly, some assume a dull color, and others only enough to show their outline. It is, therefore, a very valuable agent. Many of the larger masses are shown by this stain to be built up of minute spherules differing from each other in some chemical character, and yet acting together in building these compound forms. They might be called mulberry forms, since they are composed of various little round masses which we may call the *primary spherules*. These are generally no larger than the nucleus of an ordinary epithelial cell; so small indeed that they may circulate in the blood stream

without interference, or probably pass through the glands with the usual secretions, when there is an overplus of the material to be thrown out, or in any other condition of the blood in which these chemical constituents are not retained. The phenomena presented, when viewed in this way, are of wonderful interest.

Nigrosin is a diffusive stain for much the greater part of the material, but it has one point of differentiation not made by any other stain that I have tried. There is one class of spherules in many of the specimens — not in all — that nigrosin leaves perfectly transparent. (See Figures 129 and 130.) This spherule is often larger than others, or possibly made up of many smaller spherules, none of which take the stain, and therefore are invisible. In very thin deposits, one of these is often the center of a cluster of other small spherules which take the stain. This causes them to appear as if formed around an opening. In thicker deposits these — which seem to have been the first deposited — often peep through among the darker ones, by which they are nearly covered, as tiny stars of clear light, or as larger areas of light where they have less covering. This makes nigrosin a very interesting stain. It is curious to note the greater variations in markings brought out by these different stains, and the demonstrations of differences in chemical preferences by spherules which are thrown out of the circulation together and so intimately associated. Perhaps some other persons more familiar with the modern methods of handling stains and mordants, and also with more time at command, would be able to produce other differentiations which I have not found. The differentiations mentioned are represented as well as possible in plain light and shade in the series of photomicrographs presented. They are, however, a very poor representation of what is actually seen with the microscope.

This staining of these different globulins, which make up the agglutinin of salivary calculus, is not different in theory or in what it teaches, from the staining of tissues when properly prepared, cut in fine sections and then their different parts brought into bold view by selective stains. The epithelial cells stain differently from the connective tissue cells. In each of these again, the nucleus stains differently from the body of the cell. In each case, the differential stain is a response to chemical preference — an exhibit of chemotaxis founded upon a chemical difference in the particular portion of tissue.

These selective stains of the spherules of agglutinin show conclusively that this substance is made up of a number of glob-

ulins which differ in some particular features of their chemical structure. We can not from these, select and name the globulins represented. It seems best, then, that we continue to call this mass derived from the saliva the agglutinin of calculus.

Deposits classified.

I have previously spoken of three classes of deposit. (1) Agglutinin of salivary calculus loaded with calcium salts, and of a greyish-white color; the central zone. (2) A considerable deposit of the same agglutinin, so far as I am able to determine by physical, microscopical and staining tests, which carries with it very little of calcium salts. When very fresh, this is semi-transparent or slightly greyish; the middle zone. (3) A still more scant deposit, fully transparent, which in staining tests seems to lack certain of the classes of spherules of the agglutinin present in the other two forms; the outer zone. Those spherules which stain brightly with gentian violet are missing in the outer zone. It is possible, however, that the presence of the calcium salts may so affect the staining as to be deceptive on this point. These zones are shown best in Figures 126 and 131.

These three zones are usually present in the material laid down in each paroxysm; the greyish-white deposit occupying the center of the area, surrounded by the semitransparent or middle zone, and still farther out by the transparent or outer zone. These differences are not usually very sharply defined, but grade imperceptibly into each other. The central, or greyish-white, form is never seen alone, but is surrounded by the other two. The semitransparent and the transparent zones are often seen without the greyish-white zone. Not very frequently the transparent zone is seen without either of the others.

Paroxysmal characters.

While I was studying the masses of calculus by aid of staining agents, other studies were also being carried on. One of the first determinations certainly made was that the deposit of salivary calculus is paroxysmal. With myself, and others in good health, whom I have had the opportunity to examine sufficiently for a determination, there has been no exception to this rule. Some persons, who were in a very low state of health, were regularly examined by others, and reported as having a deposit every day on plates worn. This deposit was of the semi-transparent and transparent agglutinin of calculus, but none

of the greyish-white form, containing calcium salts. These persons may have paroxysms of the deposit of the white form. This point needs further investigation.

For a considerable time the cause of these paroxysms of deposit eluded me. I instituted the most rigid scrutiny of my own actions and doings — how I was employed, how I slept and what I ate. For some time I weighed the food eaten at breakfast and dinner and took notes of my noon lunches at the restaurant.

The plan of study was not long in bringing results. It was found that palatable meals, eaten of heartily, and apparently well digested, were followed by paroxysms of deposit of salivary calculus. (See Figures 131, 132 and 133.) These meals did not produce any notable discomfort, but after studying the matter more closely, I found there was something of a heaviness and languor following such meals, but nothing more. One of my students, a jolly and rather fat fellow, expressed the matter in this way. He told me that after hearing my lecture on this point, he concluded he would try it himself, for he had to have calculus removed from his teeth very frequently. He said he "cut his meals in two in the middle." He didn't get hungry, he didn't lose flesh, he didn't have any more calculus on his teeth. "But," said he, "that isn't half the story. Before trying this out I was absolutely unable to read or study for more than one hour of an evening. I would go to sleep in spite of everything I could do. But now I can work from eight to eleven every evening and feel good all the time. No more big meals for me."

When this matter was determined, I stopped the paroxysms of deposit in my own mouth completely, except as I produced them in the study of the effect of different articles of food. Or, if I wanted a fresh deposit of calculus for study, I was able to get it. If I went to my restaurant and ordered boiled pigs' feet and sauerkraut and ate the full order served, I was sure of a flood of calculus within three hours, which might continue several hours. If I ordered the pigs' feet and sauerkraut and ate but half the order served, I had no calculus. Braised meats of any kind, with rich brown gravy, eaten heartily, usually produced a paroxysm of deposit, but if eaten more moderately produced no such effect. I can drink one glass of milk before retiring at night and rise in the morning with a perfectly clean plate. If I drink two glasses of milk before retiring, I will have a good specimen of calculus next morning. The kind of food seems to make very little difference, an excessive amount of

almost any food produces a deposit. I found that I could induce a paroxysm of deposit with almost any good nutritious food, even simple bread and butter.

One day for luncheon I ate two-thirds of an order of "baked young pig and sweet potatoes." When I returned from lunch I was more careful than usual in cleansing my plate. This was at two o'clock. During the afternoon I was busy with other things and forgot the matter. That evening a few minutes after six o'clock, I examined the plate and within these four hours, a flood of calculus had been poured out, which covered up and hid my trap completely and filled both buccal sides of my plate nearly to the cuspids. All of the central zone was almost snow white. I cleaned the plate carefully and ate my dinner. When I stopped writing at midnight the plate was found to be clean. It was also clean next morning. This paroxysm with its extraordinary amount of material had come and gone within the four hours after eating. Curiously enough, the more I study this point regarding the duration of paroxysms the shorter I find them.

Gathering calculus direct from the parotid gland.

In order to determine definitely that calculus comes into the mouth as calco-globulin, I instituted another series of experiments. Saliva was collected before it reached the mouth by the intubation of Stenson's duct. Special apparatus was designed for the purpose: a tube was passed into the duct and the saliva was collected in a test-tube, without ever having touched the tissues of the mouth. (See Figures 135 and 136.) A test of the saliva so collected, by placing it in the incubation oven, showed it to be sterile.

I selected two young men, who frequently had calculus on their teeth, and took them out to luncheon. I gave each of them a plate with two whole pigs' feet which had been boiled with cabbage, and they were invited to eat anything else they wished. The pigs' feet had been boiled until they were soft and tender. Of the two young men in the first group that I employed for this purpose, one of them ate all of his order, gnawing the bones clean. The other picked out certain bits, and did not eat more than one-fourth of the order. About two hours afterward, tubes were put into the ducts and the saliva was found to run fairly well. We will call these two men A and B. In three-quarters of an hour 8 cc. were collected from A and 6 cc. from B.

Cover-glasses for catching the deposit were placed to the bottom of the test-tubes before beginning the collection of the saliva, and remained there. As the running of the saliva continued, it was noticed that in the case of A, who ate the full amount of the order of pigs' feet, the saliva was turbid in the tube; in that of B, who ate only a part of the order, it was clear. A little later, in the saliva of A, a deposit of snow-white calco-globulin could be seen upon the cover-glass in the bottom of the tube; in that of B, toward the close of the experiment, there was some deposit on the cover-glass, but it was very slight. The saliva in the tube remained clear.

At the end of three-quarters of an hour the experiment was discontinued, believing that we had enough to make a good test. The tubes were then set away. I did not put them in the incubation oven, as the weather during the day had been 98° in my room, making the room itself an incubation oven, and it remained so through most of the night. The next morning I found the saliva clear in both tubes. In that of A, a snow-white deposit was piled up on the cover-glass — all that would lie on it. The cover-glass was ⅝ inch in diameter and was piled up fully ¼ inch high from the 8 cc. of saliva, only 7 cc. of which were above the cover-glass, and the space under the cover-glass was well packed with calco-globulin. In the tube from B there was a very good microscopic specimen of calco-globulin on the cover-glass, but no great accumulation.

Any one may make the tubes and conduct a similar series of experiments. The ducts should be examined in each case before anything else has been done. In quite a number of persons, Stenson's duct was found to be so small that a tube, the lumen of which was large enough to serve the purpose well in collecting, could not be passed into it. In others, the duct was so tortuous that it was exceedingly difficult to follow it with a tube to sufficient depth for the tube to hold well. Many difficulties arise which must be overcome. Persons must be selected who are known to have considerable deposits of calculus, and it should be determined that the tubes may be passed into the ducts. Then they should eat a heavy meal of food that is highly nutritious and easily digested, for the purpose of arousing a paroxysm. The tube should be inserted about one and a half hours after the meal and observed carefully in order to catch the paroxysm at its height, for it is not uncommon for these paroxysms, with a great flow of calculus, to be over in half an hour after they begin.

The gathering of calculus in this way marks a step in advance in our study of this deposit, one that will stand for all time, showing whence comes the calculus that we find upon the teeth. In the endeavor to collect calculus in this way, a great many failures will occur. We must not take persons who have never been known to have calculus on their teeth, and expect to find accumulations in the saliva drawn. We must not expect to succeed in every case, even though we have made the very best possible selection of a subject. If a person's digestion happens not to be good at the time, the food may arouse no flow of calculus, because if the food is not well digested, there will be no excess of calco-globulin.

Deposits of hard salivary calculus are occasionally found in the ducts of the salivary glands, the nidus for such deposits usually being some foreign substance, such as a splinter of a wooden toothpick, which has been accidentally passed into the duct. I have seen several such calculi of considerable size. Figures 140, 141 and 142 are actual size reproductions of photographs of calculi removed from the ducts of the salivary glands. There is also shown in Figure 143 a tremendous deposit which was found in a kidney of a cadaver in the anatomical laboratory.

Globulin.

A globulin is any one of a class of albuminous proteid compounds insoluble in water or alcohol, but soluble in weak solutions of the neutral salts. The animal globulins include fibrinogen, serum globulin or paraglobulin, globin, myosinogen, crystallin, and vitellin. This definition was written after consulting a number of medical and general dictionaries, but is not quite like any one of them. I have been unable to determine definitely why this term globulin has been applied to these products, but must suppose that some one has seen the spherical forms, and consequently applied the term globulin.

Globulin is recognized by physiological chemists as a highly nutrient material held in the blood, body juices and flesh, in readiness for use in tissue metabolism of the constructive type; a nutrient material in excess of immediate needs, but ready for immediate use. In this respect the globulins are very important in the nutritional processes. As previously stated, in this method of procedure the agglutinin of calculus is found to respond to the usual tests for globulin.

FIG. 131.

FIG. 131. The appearance given by an accumulation rapidly formed (within four hours), stained with gentian violet, after having been coagulated by phenol, 5 per cent, solution in water, and the uncombined phenol carefully dissolved out by repeated washing. The lower right-hand corner of the picture was in a corner of the frame.

It will be noticed in this that certain of the smaller spherules stain more prominently than others, and that the larger spherules are agglomerations of the smaller spherules. This specimen was perfectly transparent before coagulation with phenol. At certain points the accumulation was too thick for photography.

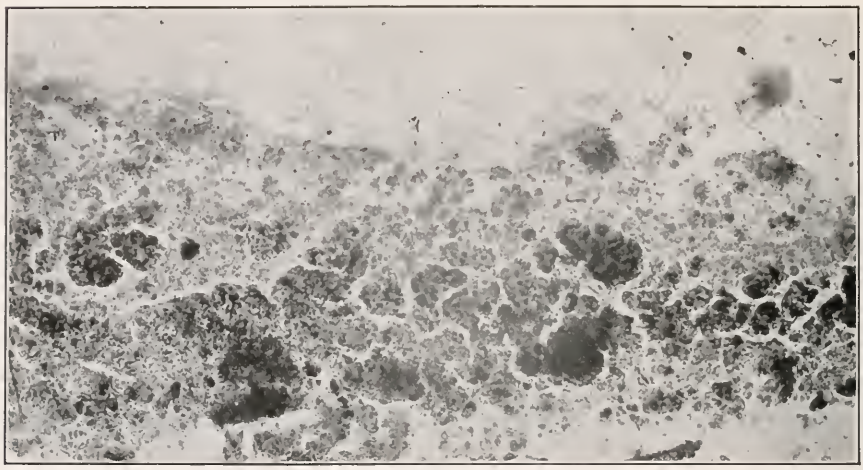

FIG. 132.

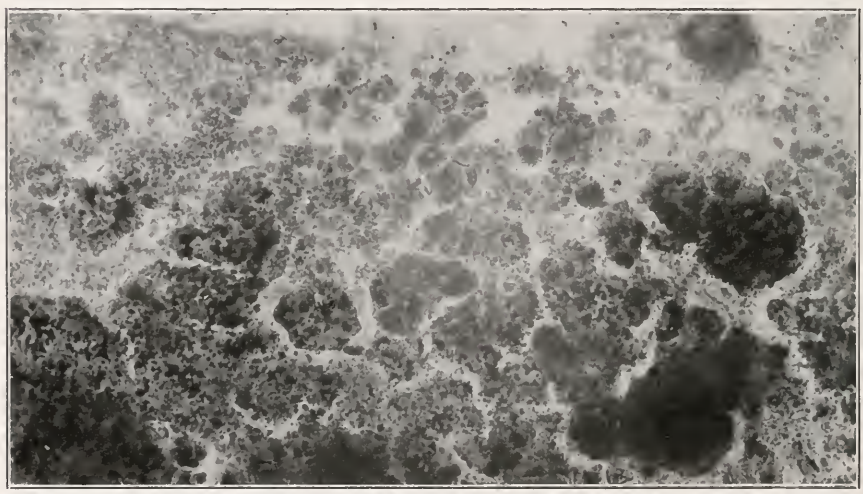

FIG. 133.

FIG. 132. Appearance given by an accumulation forming in about ten hours, stained by gentian violet after it had been exposed to 4 per cent formalin for one hour, and repeatedly washed to remove uncombined formalin. It remained in the staining solution twenty-four hours, and was washed in absolute alcohol, which was repeatedly changed, and much of the time kept in motion for about two hours.

This specimen gives a beautiful appearance when seen in the microscope, but as the stain is a bright blue with gradations of the intensity of color in the different primary spherules, it is only imperfectly represented by photography. The conglomerate structure can, however, be made out fairly well.

FIG. 133. From the same specimen shown in Figure 132, showing only the central portion of the field. In this a multitude of fine spherules that take the stain sharply appear in the make-up of the large spherules. In some specimens the masses are made up of primary spherules that take the stain differently, showing distinct chemical differences in these primary spherules that join in making up the larger compound forms. Thus far this is well shown only by the gentian violet stain, following formalin as a mordant. Other stains may yet be found that will make these selections and be better for photographing.

AGGLUTININ OF SALIVARY CALCULUS.

As yet the agglutinin of salivary calculus has been but partially studied. I have given attention for the most part to the physical phenomena rather than the chemical qualities. At present the former are much the more important qualities to be made out. When a substance which is well known is identified, that is sufficient. What it does in the new position in which it is discovered becomes the important question. I have adopted the term *agglutinin* or *agglutinin of calculus* because we should have a distinctive name for the substance as it appears in connection with deposits of calculus, to distinguish that form from every other, no matter what it may be chemically. That it comes into the mouth with the saliva there is no question.

Agglutinin, as we find it deposited in the mouth, when taken up in the fresh state — as a deposit discovered while being laid down during the day, or one discovered in the morning, having been laid down during the night — is so much heavier than water that it will sink at once. It will do the same in fresh saliva. It is probably not in actual solution in the saliva at all, but is distributed in the form of very small primary spherules. From these the larger masses seen in deposits are built up. (See Figures 123 to 128.) It now seems that there can be no question that it is deposited directly from the saliva almost immediately upon entering the mouth. The bulk of the material thrown out is carried away with the saliva and is never seen.

It requires especially favorable conditions for it to become deposited in the mouth, upon the teeth, or even upon hard substances, as plates, worn in the mouth. As will be mentioned later, it was found that deposits did not occur on a cover-glass held in place with a gold frame, unless the frame was made with an angle or rough edge which would give opportunity for the first deposit to occur. A study of the deposits occurring on artificial dentures shows that the beginnings are also in sheltered places. This should lead to the making of more perfect denture forms so that deposits will not occur.

CONSISTENCY OF THE SPHERULES. These spherules are very soft when gathered with the saliva and allowed to fall upon a cover-glass fixed in the bottom of a test-tube to catch them. They will accumulate and pile up on the cover-glass until after a time they will roll down the sides of the pile and over the edge of the glass. A little disturbance will cause them to roll off as they accumulate upon the glass. If the tube is kept still, how-

ever, they will, in the course of a few hours, become attached to the glass as with a very soft, sticky wax. (See Figure 137.) This waxy consistency in the spherules is very characteristic, for it is by this that they take hold and adhere to hard substances, which form nidi for the gathering of calculus, as the teeth, plates and various hard substances placed in the mouth, or any object that is put in the urinary bladder and serves as a nidus. Once I found a small pen-knife in the urinary bladder of a girl, which had acted as a nidus and was encrusted with calculus. Anything of this kind, or sometimes a group of dead cells, seems to act as a nidus. The peculiar characteristics of the globulin spherule are such as to cause it to cohere in this way, one of its sides flattening down upon hard substances; hence it gathers on teeth, plates, etc.

From this description it will be noted that the original spherule, as eliminated from the salivary glands, or other sources, is a very soft mass that will not stand alone and preserve its rotundity, if laid upon a flat surface, but will slowly come to stick fast, and change its form, presenting a flat side upon the substance to which it sticks. Others, each falling upon a layer of these, will stick to them, and so on until a very considerable accumulation may be built up. The building of these depends very largely upon the sticky character of the spherules.

We may get special preparations in the following way: While the saliva is still running, place the point of a small glass pipette into the liquid which has accumulated in the test-tube, and draw some of it into the tube of the pipette. Place the pipette so that it will be perpendicular with the point on a cover-glass, and let it stand for some little time to permit the spherules to sink to the point. Then slight pressure upon the rubber bulb will cause a little of the liquid to issue, in the form of a drop, on the cover-glass. Preparation should have been made beforehand, and this should now be placed in a moist chamber, to prevent drying, and should remain there several hours, in order that the globulin may become stuck fast to the cover-glass. Then it can be passed into alcohol, or into staining fluids, and handled so as to make the preparation such as is desired for mounting.

To gain some idea of the consistency of these spherules, one may imagine spherules made of a soft wax, warmed until it becomes so sticky that the spherules will first cohere and then gradually coalesce. Each will sink in among the rest and gradually lose its identity. In settling down, there is the tendency to form larger rounded masses. This is apparent on examining

the material stained on cover-glasses. Some of these are in the form of more or less flattened spheres.

As to softness, the spherules which contain a large amount of calcium salts seem to be just as soft, or if anything a little softer, than those spherules which contain no calcium salts, so far as the eye can detect.

Globulin and salts inseparable. The spherules containing calcium salts are in no sense hard calco-globulin, but they seem to be chemically combined nevertheless. The one can not be dissolved without dissolving the other. It may be possible to dissolve the globulin and precipitate the salts by the addition of chemicals, but in every attempt which I have made to isolate the salts from the globulin without adding chemicals, the whole body has been dissolved, salts and globulin. I tried hot water, and after five days, six hours per day, at a temperature of 206° F. — 209 is the boiling point at our altitude, barometer 30 (sea level) — a solution was practically complete, only a very scant waxy deposit remained. Practically the whole material became hydrolized and on evaporation formed crystals not resembling the crystals of calcium phosphate. No precipitate of calcium salts was discovered; no amount of mechanical agitation seems to succeed in shaking out a precipitate of calcium salts from the soft deposits of calculus.

Deposits during illness.

I have also discovered another feature of this deposit which is very interesting, though not well worked out. A dentist, who was interested with me in the study of this subject, reported that the full artificial dentures worn by his wife, who was a paralytic, were covered with a thin veil of the transparent zone of salivary calculus every day. It was necessary for him to clean these dentures, as she could not do so, and he examined them carefully each day for a considerable time. If the cleaning was neglected for a few days, the deposit was considerably thickened and became of a brownish color and the cheese-like consistence. In this way she seemed to be losing by leakage through the secretion, so to speak, the scant supply of globulin which was formed by her very imperfect digestion of food.

In 1912 I spent three summer months on my farm under what seemed to be ideal conditions for recovery from a neuritis. I slept in a bungalow, the walls of which were made of screens, except a dressing-room and a bathroom. Practically, we had our meals, lived and slept in the open air. I also spent most of

the time of daylight in the woods directing some laborers. I was taking about as much out-door exercise as my condition would allow.

During this time I had much more deposit of calculus than when at home pursuing my usual work. That is, a deposit seemed more easily aroused. This was a surprise, and I watched it very carefully and made the best comparison I could with my consumption of food. While this influenced it sharply, a deposit was much more easily aroused than formerly. After my return to my usual employments, this condition ceased. I do not know why.

This circumstance, taken with what we see among our patients, seems to confirm the supposition that there are some systemic conditions which favor the deposit of calculus, besides the quantity of food taken.

In this we must remember also, that there are many people who never have a deposit of calculus upon their teeth. Yet, so far as we can see, their habits of living are the same as those of other people. They are not different in physical qualities nor in degree of general health. In the consideration of the deposit of calculus this must not be overlooked. It speaks very emphatically of a calculus dyscrasia of which we have as yet no tangible idea.

A few times I have seen the deposit of agglutinin in the clear form so abundant that while it did not appear to the eye in its clear, transparent freshness, it would hide the plate completely when coagulated by boiling water, in which it would be whitened and become opaque. A deposit of this extent is evidently quite rare, but I have seen it several times. It has occurred a few times on my own plate, in conjunction, however, with a whiter deposit about the openings of the ducts of the glands. This, with myself, has always occurred during some illness. (See Figures 138 and 139.)

In passing, I may mention the fact that not infrequently calculus is found on bullets and other metallic substances lodged in the flesh, which have remained for some time. This calculus as I have examined it, particularly on lead, has been softer than the calculus formed in the saliva or in the urinary bladder. In some specimens it has been so soft that I could crumble it with my fingers, and yet there was no doubt whatever of its character. This shows plainly that calculus may also be deposited from serum exuding from the tissues, and is therefore carried by the blood stream.

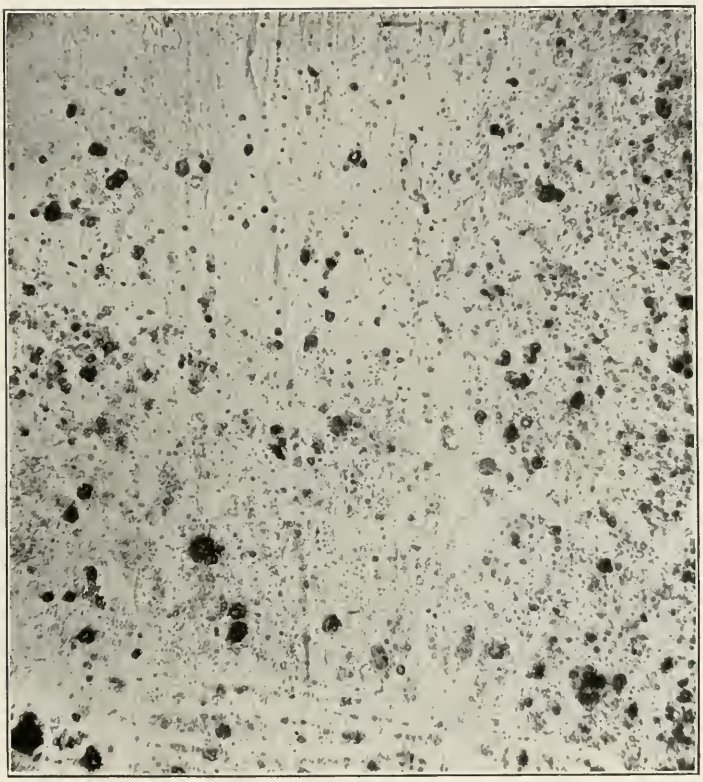

Fig. 134.

Fig. 134. A slight but very diffuse deposit occurring slowly at the ending of a paroxysm. There is a sprinkling of spherules that take the stain sharply, among many fine spherules that do not, and a considerable number of small circles scattered over the field formed by accumulations around spherules which do not take the stain.

FIG. 135.

FIG. 135. Intubation of Stenson's duct for the purpose of collecting the saliva as it comes from the parotid gland. A silk ligature, previously tied around the brass tube, is tied to a bicuspid tooth to hold the tube in the duct. A brass wire is so bent as to hold a disk crosswise of the test-tube. This disk supports a cover-glass. (See Figure 136.) As the saliva, which drops from the brass tube, accumulates in the test tube, it will be cloudy if it contains much calco-globulin, and the spherules will gradually settle and collect upon the cover-glass. (See Figure 137.)

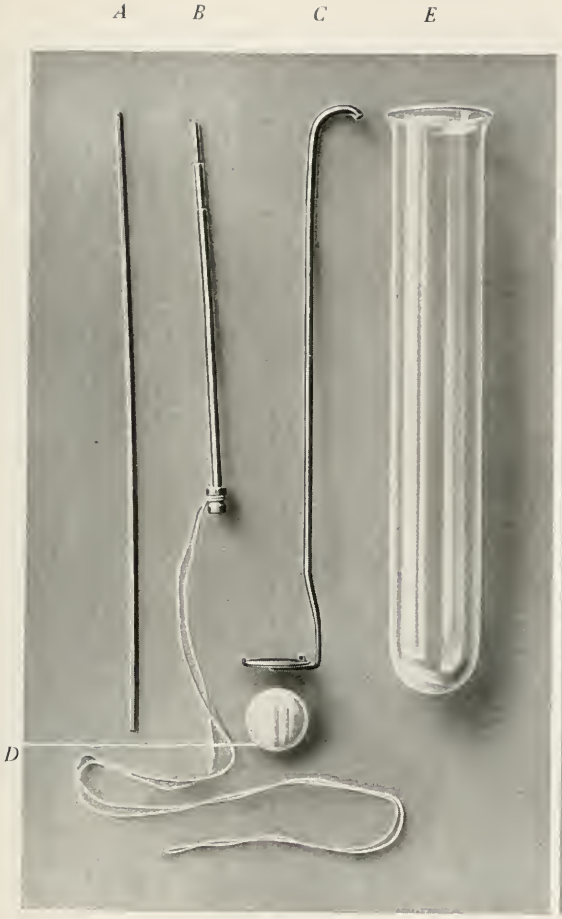

FIG. 136.

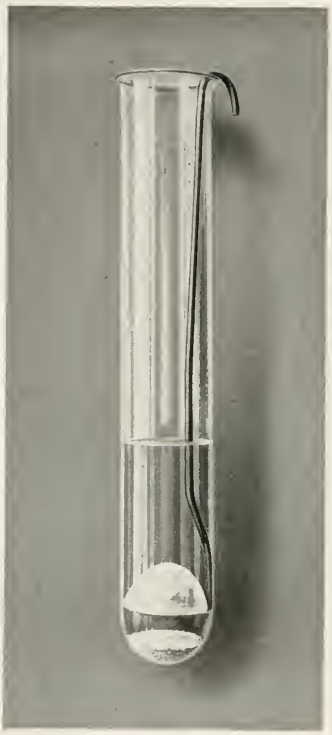

FIG. 137.

FIG. 136. Intubation set for collecting saliva direct from Stenson's duct. A, Canula for intubation tube. B, Tube with silk ligature attached. This ligature is tied to a bicuspid tooth after the tube is in the duct, preventing it from slipping out. C, Wire with disk attached, to hold cover-glass, D, in horizontal position in test-tube. E, Test-tube for collecting saliva.

FIG. 137. Representation of the test-tube with saliva containing calco-globulin. The saliva is at first cloudy and gradually clears as the spherules settle upon the cover-glass. In excessive paroxysms the spherules will pile up on the cover-glass until it will hold no more and then roll off and accumulate in the bottom of the test-tube.

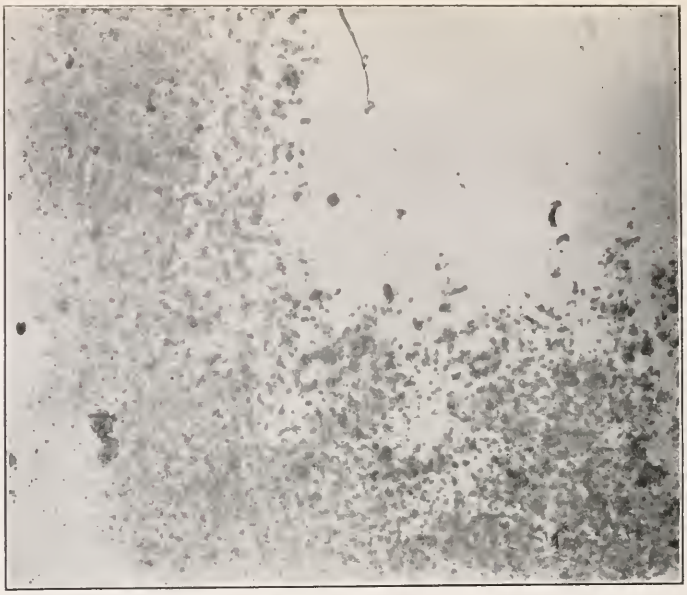

FIG. 138.

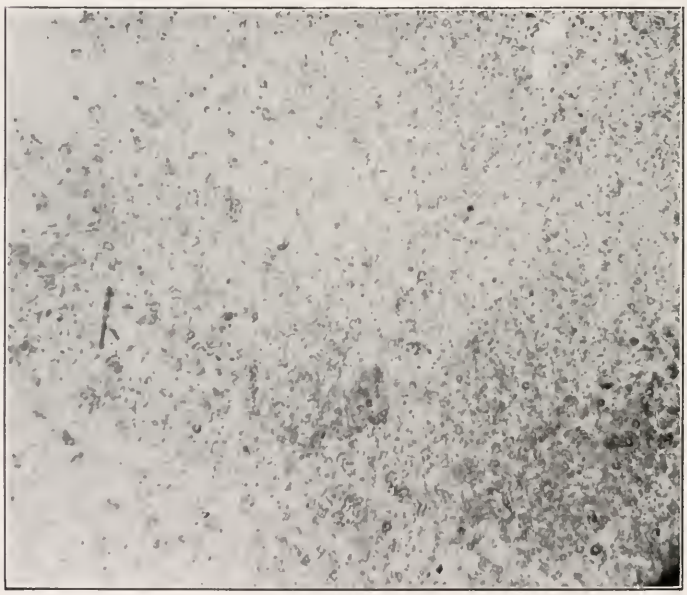

FIG. 139.

FIG. 138. This is a deposit of a peculiar type that seems not to contain calcium salts, but will form a cheese-like accumulation on the teeth or on dentures. When cleaning is neglected for a few days, this becomes too stiff to be removed with a brush, but may be scraped off with the finger nail. This form has been observed oftenest on dentures of persons whose nutritive powers are very low. It is very persistent in some confirmed paralytics. It has not, however, been sufficiently studied. Nigrosin stain.

FIG. 139. This shows a deposit of finer spherules of a type of material similar to that shown in Figure 138. Nigrosin stain.

It has been with some difficulty that I have arrived at the conclusion that so important a nutritional substance as the globulins are shed out with the secretions and become a principal factor in the production of such a substance as salivary calculus, but the facts recited force this conclusion.

I do not know how many persons in very poor physical condition are having such deposits as have been mentioned, or are losing the globulin from their blood in this way. My finding, however, is a strong suggestion that this may be a mode of draining away the nutritive power of the blood in such conditions.

Chemistry of the deposits.

The chemistry of these deposits needs closer investigation. It seems now that the following supposition may be found correct: The greyish-white, or central zone, of the deposit is composed, in the main, of a semifluid calco-globulin, but has mixed with it more or less primary globules not containing calcium salts. The mixture of calco-globulin within the mass becomes thinner as we proceed from the center outward, until at some certain point we have globulin only, as found in the outer or transparent zone. Physical examination of the mass denotes this change and also the observation of the hardening process shows the two parts to act differently. The white portion will partly decompose, leaving a stony, hard calculus, while the clear portions will harden first to the consistence, but not the color, of cheese, and finally break up and disappear. It is at first slightly yellow, almost transparent, and becomes opaque and of a darker color as it grows older. Whether or not the lactic acid formed in the mouth has any important action in the removing of calcium salts from the more thinly scattered deposits, and, in this way preventing the hardening, may be mentioned as one of the undefined chemical problems of minor importance.

Whatever the full truth may be as revealed by future discovery, the fact will remain that in the ordinary paroxysmal deposit of persons in good, or fair health, the three zones of deposit will appear, varying widely in the proportions of each. The deposit close about the opening of the ducts, the central zone, carries the bulk of the calcium salts.

A condition of general deposit of calculus, about practically all of the natural teeth, is, however, occasionally seen. These are generally neglected cases in which no effort has been made to keep the mouth clean. In such cases, it has seemed to me

that there is established a condition of putrefactive decomposition which acts as a preventive of the formation of lactic acid, or neutralizes the lactic acid formed. The study of these changes presents the opportunity for some chemist to do an important work.

Hardening of salivary calculus. The chemistry of the hardening of salivary calculus seems to have no literature. That it is deposited in a soft condition and slowly becomes hard, is stated by many writers. With that statement the subject is dropped.

I have only begun some investigation of this process from the physical side. The hardening is found to be accompanied by putrefactive decomposition of the agglutinin during which the transparent portions, as well as the white zone, first become opaque and yellow, diminishing very much in bulk. If the specimen is placed in clear water, this soon becomes clouded with a growth of micro-organisms. Then the changes seem to go on much slower, although apparently of the same character, but the change to the hard, stone-like form, such as is found upon the teeth, does not occur. Ordinarily it is very difficult to follow this process with any degree of accuracy in the mouths of patients. It is also proving to be a difficult matter to contrive artificial conditions which will serve much better. It seems probable now that the hardening can not be studied by any artificial method out of the natural position in the mouth.

I have placed a number of specimens in water at room temperature and some at body temperature. The results were similar in both. Within a short time the water became white from the growth of micro-organisms, which continued for a week or two. Then the fluid became clear. During this time the deposit lost perhaps three-fourths of its bulk. What remained had not become hard. One very heavy white deposit was placed, plate and all, in clean water and closed with a close-fitting lid, but not sealed. At the end of four months what remained of the mass was of a yellow color and so soft that I could pick it to pieces with the small end of an ordinary wood toothpick, finding only an almost imperceptible resistance. Still more recently, I have packed masses of the fresh soft deposit on a cover-glass and suspended it by a clutch fastened in the cork, and corked it tightly in a short glass tube one inch in diameter. In the bottom of this tube was placed a piece of cotton with as much water as it would hold without being liable to run. This specimen was taken from a very thick paper-white

deposit. At the end of the second day it was much too hard to brush away; in five days, yellow and softer. It did not harden as is the mouth.

KNOWLEDGE OF HARDENING BASIS FOR PROPHYLACTIC TEACHING. The meaning of a wider knowledge of the deposit of salivary calculus in its relation to prophylactic work can hardly be reckoned. This should stand as the basis of much of the popular teaching of prophylaxis. Our people should learn the facts in brief, substantial statements, accompanied with equally definite statements as to how to deal with them. Deposits of calculus of any degree removed twice per day — morning and evening — will do no harm whatever to the gingivæ or to the teeth. All people, with but a few exceptions, should be able to so conduct their food habits that no deposits of calculus would occur; or, by cleaning after each meal, calculus may be prevented from doing harm to the tissues.

EXPLANATORY SUPPOSITION.

The facts here stated give origin to some suppositions that seem necessary to a fuller explanation of the method of the instigation of the paroxysms of deposit of this material.

The most rational supposition which has come to my mind is this: With a very full meal of highly nutritious food, with the alimentary apparatus in good condition, much more nutrient material is thrown into the blood, and more of the globulins are formed than are necessary or can be used. At a certain, or, perhaps, variable overaccumulation of these, the excess is shed out with the secretions and excretions, and then a proper equilibrium is again established. This marks the rise and decline of the paroxysm. This is at least a thinkable explanation of a process, as yet hidden, which seems to meet the now known facts and affords a resting-place for thought regarding the most common form of paroxysm which begins, rises to its flood, abates and ceases within from one to three or four hours. It is rather rare that I have seen closely watched paroxysms of much longer duration. I have seen a number which did not continue for more than half an hour. A much closer watch than had at first been made, has convinced me that paroxysms lasting two or three days, as I have heretofore stated, have been two or more paroxysms in close succession instead of a single paroxysm, as I had supposed. This statement has in view persons in fairly robust health.

In order to keep this closer watch, it has become necessary to clean off some portion of surface receiving deposit, or a certain part of the trap itself, every hour, or even every half hour, until no more deposit is discoverable.

In persons in chronic ill health, and especially those whose condition is that of marked malnutrition, an almost constant slow deposit of agglutinin seems to occur. This may not carry with it any deposit of calcium salts. In that case it does not harden as do other deposits, but settles into a curd-like mass which strongly resists removal with the brush, but may readily be scraped away. This form of deposit never becomes very hard. The material, however, gives the same appearance in stained specimens as that containing calcium salts. This form of deposit of agglutinin sometimes creates a very foul condition of the plate and mouth through the putrefactive decomposition which occurs in the mass. A low form of inflammation of the soft tissues occurs wherever this decomposing material is in contact with them.

In the class of cases just mentioned, the opportunity for the careful daily observation of many persons has not occurred to me, but daily examinations have been made by others and reported to me. From what I have learned, it would seem that the nutritional process is so low that much of the small amount of the globulins formed are leaking away with the secretions and excretions, and these are being lost. This condition acts to intensify other diseased conditions, or may occasionally form the basis of a type of wasting disease.

Calco-globulin in other secretions.

I have not yet examined other secretions than the saliva for globulin. The literature gives but little information on the subject. It seems to me probable that during certain hours after heavy meals other secretions will be loaded with globulin the same as the saliva. These would give a deposit of calculus if a nidus happened to be present, as occurs in the urinary and gall bladders and at some other points.

Calculus in the urinary bladder has a wide literature, but at present this does not help us. While we find many matters dependent upon these deposits very ably and fully discussed, the intrinsic part of the subject seems to have had no very careful examination.

Fig. 140.

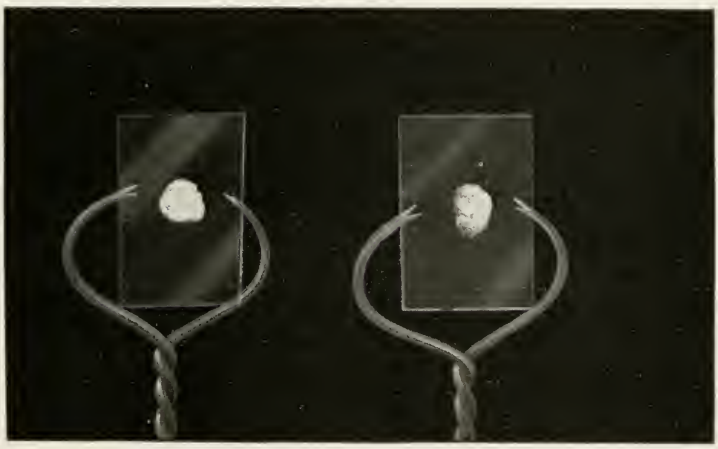

Fig. 141. Fig. 142.

Figs. 140, 141, 142. Reproductions of salivary calculi removed from ducts of salivary glands. Actual sizes. Specimens from Northwestern University Dental Museum. Figure 140 presented by Dr. Edward C. Tyler, Traverse City, Mich. Figure 141 by Dr. W. R. Wolf, Parsons, Kan. Figure 142 by Dr. P. A. Pyper, Pontiac, Ill. It should be particularly noted that these deposits occurred from the saliva before it reached the mouth.

Fig. 143.

Fig. 143. An enormous deposit of calculus in a human kidney. Actual size. This specimen was found in a cadaver in the anatomical laboratory of Northwestern University Dental School, by Dr. William Bebb, curator of the Museum. It seems probable that renal and all other calculi are closely related to the salivary calculi, and that the cause of all is the same.

Globulin urea, however, has something of a literature. I quote the following from a paper which I read before the Chicago Dental Society in January, 1912.*

"In general medicine it seems probable that much useful information of importance in diagnosis may be derived from a study of the elimination of the globulins. How and where, for the most part will be determined by trial, or in other words, a general study of the subject as it appears in the secretions, and especially in the excretions. Already globulinuria has a considerable literature, though the knowledge of it seems to be rather indefinite.

"In an article entitled 'Euglobulin Reaction in Urine,' by Arthur R. Elliott of Chicago, in the Illinois Medical Journal for November, 1911, p. 520, the progress of this literature is cited. There is also a discussion of the difficulties of diagnosis between albuminuria and euglobulinuria, a matter which is very liable to confusion by the close resemblance of the chemical reactions of the globulins to albumin.

"From what has been said of the deposit of globulins in the form of agglutinin of calculus, one would expect the appearance of the globulins in urine to be transient. This, in fact, is what seems to occur, and these have been referred to as innocent albuminurias. This article by Dr. Elliott is worthy of study for both its citation of authorities and its discussion of the means of differential diagnosis now known.

"If the physician could command the time to learn to distinguish readily the paroxysms of the deposit in the mouth, he would probably find a coincidence between this and the innocent albuminurias, and that each is an expression of the same systemic condition. It is possible that the careful comparative study of the saliva and urine may lead to clearer definitions and simplify the means of division of the grave and the innocent albuminurias.

"But are these repeated paroxysms of elimination of globulins innocent of injury to health? May they not have a causative relation to some grave conditions, or a harmful relation to more or less grave conditions now not fully understood? It opens up a very wide field for question and investigation.

"The examination of the saliva and other secretions, in conjunction with the urine, seems to be demanded."

Dr. Henry H. Burchard makes a quotation in his paper on

* "Deposit of Salivary Calculus," by G. V. Black, Dental Review, Vol. 26, 1912, p. 337.

the Origin of Salivary Calculus, Dental Cosmos, 1895, p. 828, from a conversation with Dr. E. C. Kirk, in which Dr. Kirk is quoted as saying: "I believe all of these calculary deposits will be found to belong to one great order. That salivary calculi will be found to be one group of several chemical bodies which are formed by the precipitation of lime salts in colloid media, and this is the common factor in the formation of calculi in general. That about a nidus these substances will be deposited, or form in some definite manner; that they are more than mere agglomerations of lime salts with extraneous matter; that they resemble calco-globulin more than they do mere cemented precipitates, and I believe all calculi will have a family similarity in general structure, no matter in what part of the body they are found."

In this conversation, as reported here, Dr. Kirk's remarks were probably much wiser than the facts then known would seem to justify. The sections of calculus do not reveal a material that seems to be in any wise akin to that which we find in teeth, in calco-spherites in the dental pulp, or in the phleboliths found in the veins, or in those tiny globules thrown down from albumen solutions which contain calcium salts. It will be recognized that this material appears in a very different physical state from the calco-spherites deposited from colloid material as reported by Rainey and others. In the consideration of calcification within the pulp chamber I refer to the very interesting work of Rainey and Ord and show two illustrations of artificially formed calco-spherites. (See Figures 338 and 339.)

My recent search for the actual precipitate, so long regarded as the basis of this deposit, has failed entirely to show the occurrence of any such thing. Neither have I been able to isolate a precipitate of calcium salts by any means I have yet tried. When I have dissolved the globulin, the calcium has also dissolved.

Conclusion.

I am slowly, by each successive step, being driven to the conclusion that the thought of a precipitate of calcium salts from the saliva so long held, by myself and others, has been a myth. It seems now that calculus comes into the mouth as a finely divided calco-globulin which collects in masses on hard substances and is finally, with the decomposition of much of the colloid elements, hardened into stony calculus.

GINGIVITIS AND PERICEMENTITIS DUE TO DEPOSITS OF SALIVARY CALCULUS

ILLUSTRATIONS: FIGURES 144-170.

The injurious effect of the deposit of salivary calculus upon the teeth has been known since the earliest historical times. It has always been regarded as a deposit from the saliva. The calculus is deposited upon the teeth, never upon the mucous membranes or other soft parts. It is, however, the soft tissues which are injured — not the teeth themselves, except as they lose their soft tissue and bony investment. Being deposited upon the teeth, the calculus, having become hard, impinges upon the soft tissues and causes them to become inflamed and red, to bleed easily, and to become involved in suppurative processes.

Gingivitis. Beginnings and early progress of deposit.

The place of first deposit is usually on the buccal surfaces of the molars, or the lingual surfaces of the lower incisors. In both localities the deposit is close to the margin, or crest, of the free gingivæ and appears first where there is a blunting or thickening of the free gingivæ, forming a little shelf which invites the lodgment. (See Figure 144.) In many cases, however, the deposit spreads from these points and may include all of the teeth. When the deposit begins at the crest of the gingivæ, the tendency is to grow in thickness and to spread in every direction upon any parts of the surface of the tooth or teeth that are not kept clean by the rubbing of food over them in mastication, or by artificial cleaning. As this goes on, from month to month, the deposit impinges more and more upon the crests of the free gingivæ and causes them to become inflamed and bleed easily, as mentioned. As this deposit continues, the gingivæ become thickened and shortened very slowly. This greater thickness of the tissue gives a broader shelf for the lodgment of more calculus, and a broader covering is deposited over the inflamed tissues. In studying ground sections of calculus with the microscope, we find the layers of deposit as it occurs, lapping in under the thickened lower portion of the deposit, and between it and the remaining soft tissue. This is fairly well shown in Figure

163. In occasional cases, the deposit will be thicker than the thickness of the gingivæ, and will overlap the lingual or labial surface of this tissue. This is most frequently seen to the lingual of the lower incisors, but may occur to the buccal of the molars, or elsewhere. The soft tissue will often remain intact for a considerable time under this overlapping calculus. (See Figures 152, 153 and 154.)

Suppuration. The irritation of the investing tissue, caused by the presence of the deposit, and the covering of the deposit itself, offer opportunity for collection and growth of the bacteria of the mouth. Therefore, suppuration of the soft tissue, in contact with the deposit, occurs from time to time, destroying parts of the tissue, and this gives opportunity for the deposit of more calculus in the space gained. This goes on, very slowly as the rule, until the free gingivæ are destroyed and the deeper tissues are reached. Then these are involved by the suppurative process as cases progress; the bone of the alveolar process, the peridental membrane and the gum tissue all being destroyed. The products of suppuration and decomposition will often make the breath very foul.

Pericementitis. Destruction of the deeper tissues.

If not artificially removed, the encroachment of the calculus goes deeper and deeper, involving the crest of the alveolar process and the adjacent soft tissue. Indeed, the absorption of bone in such areas of inflamed tissue is quickly accomplished. (See Figures 144, 145, 146 and 147.) On examining this tissue immediately after the removal of the calculus, soft granulation tissue only will be seen. A sharp steel probe will, however, show but a slight covering of granulation tissue over the shortened and apparently thickened stub of the partly absorbed alveolar process. In this way, the bony alveolar wall is destroyed, little by little, from month to month, as more and more calculus is added, going deeper and deeper along the root of the tooth to which it clings. All of the investing tissue, soft and hard, is destroyed as this progresses. (See Figures 155 to 167.)

Attachment of peridental membrane to root maintained to level of soft tissue remaining. No matter what the extent of the injury, the attachment of the peridental membrane to the root is usually maintained to the level of the soft tissue remaining. (See Figures 144, 145, 146 and 147.) Any considerable accumulation may be broken away from the tooth with suitable instruments, and give a clear view of the process of destruction.

FIG. 144.

FIG. 145.

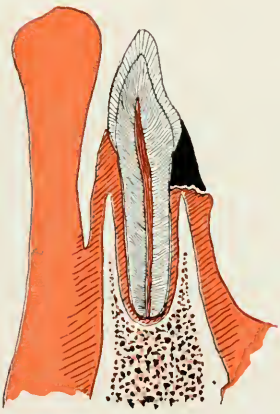

FIG. 146

FIG. 147.

FIGS. 144, 145, 146, 147. Drawings to illustrate the progressive destruction of the investing tissues caused by deposits of salivary calculus.

FIG. 144 shows a slight deposit on the lingual surface of a lower incisor which has caused a gingivitis only, not having progressed far enough to involve the attachment of the peridental membrane to the cementum.

FIG. 145 shows a similar slight deposit on the buccal surface of an upper molar.

FIG. 146 shows a more extensive accumulation on the lingual of a lower incisor than that shown in Figure 144. It will be noticed that the gingival line of the tooth has been passed, and the deposit has almost reached the crest of the bone.

FIG. 147 shows a still greater destruction, including also the labial tissues.

In all of these it will be noticed that all of the investing tissues — gingivæ, peridental membrane, bone and gum — are destroyed on a line practically horizontal to the long axis of the root, and pockets alongside the root are not formed.

FIG. 148. FIG. 149. FIG. 150. FIG. 151.

FIG 152. FIG. 153. FIG. 154.

FIGS. 148, 149, 150, 151. Teeth of normal form showing extensive deposits of salivary calculus. Specimens from Northwestern University Dental Museum.

FIGS. 152, 153, 154. Lower incisor and cuspids with deposits of salivary calculus which overlapped the gum tissue. Specimens from Northwestern University Dental Museum.

Fig. 155. Fig. 156.

Fig. 157. Fig. 158.

Figs. 155, 156. Labial and lingual views of a lower incisor tooth entirely enveloped, except portion of crown, by deposit of salivary calculus. No portion whatever of the root can be seen. Specimen from Northwestern University Dental Museum, presented by Dr. Herbert S. Merdock, Springer, N. M.

Figs. 157, 158. Labial and lingual views of four lower incisors the roots of which are enveloped by deposits of salivary calculus. The movements of these teeth prevented the deposits on the respective roots from uniting. The proximal surfaces of the deposits are worn smooth from the labio-lingual movement of the teeth. Specimens from Northwestern University Dental Museum.

FIG. 159. FIG. 160.

FIG. 161. FIG. 162.

FIGS. 159, 160. Mesial and buccal views of an upper second molar with a large deposit of salivary calculus which did not destroy the tissue immediately adjacent to the root above the gingival line of the tooth, although only a little was spared. This is an unusual form of the deposit. Specimen from Northwestern University Dental Museum, presented by Dr. Arthur B. Freeman, Chicago.

FIGS. 161, 162. Two views of an upper first molar with an enormous deposit of salivary calculus attached. Specimen from Northwestern University Dental Museum.

FIG. 163.

FIG. 163. Photomicrograph of a ground section of a tooth with a deposit of salivary calculus attached to the root. The section is so thin that the structure of the dentin is not clear, although the gingival portion of the enamel may be seen at the top of the illustration. Something of the lamination of the deposit as it was gradually built may be seen. Specimen ground on the author's special grinding machine described in the Appendix in this book.

FIG. 164.

FIG. 165.

FIG. 166.

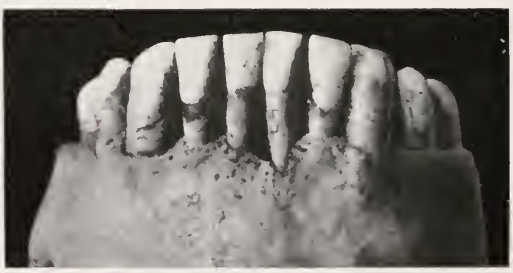

FIG. 167.

FIGS. 164, 165, 166, 167. Illustrations showing the destruction of the investing tissues by deposits of salivary calculus.

FIGS. 164 AND 165 are reproductions of radiographs showing extensive destruction of the alveolar process.

FIG. 166 is from a plaster cast of a case in which the deposit was nearly as extensive on the labial as on the lingual of the lower incisors. In this case the septal tissues were destroyed by the use of a wooden toothpick, rather than by deposits.

FIG. 167 is from a skull. This shows the destruction of the bone, which is especially deep between the central incisors. Specimen from Northwestern University Dental Museum.

Fig. 168. Fig. 169.

Figs. 168, 169. An upper and a lower plate with very heavy deposits of salivary calculus. On both of these the greatest thickness of the accumulation is about half an inch. Specimens from Northwestern University Dental Museum.

FIG. 170.

FIG. 170. A denture showing a deposit laid down in a single paroxysm. A portion of the soft accumulation was dislodged at the next meal after the deposit occurred, as shown by the spot above the position of the second molar.

The clean, white tooth crown will stand up in the midst of an inflamed, red and bleeding tissue, often showing the naked gingival line where formerly the soft tissue was attached. In many cases all of both the free gingivæ and the body of the gingivæ will have been destroyed. The rule is that pockets are not formed alongside the roots, although some detachment may occur in the more advanced cases. This is markedly different from the process by which the investing tissues are destroyed in chronic suppurative pericementitis, as will be described later.

Pain and soreness. During the early progress of this disease there is little or no pain. The teeth may become more or less tender in mastication, and the effective work of the teeth in chewing food, also the cleaning which occurs as a result of vigorous chewing, will be diminished, giving additional opportunity for the accumulation of deposits and a corresponding increase of the inflammation. It is only toward the later stages that teeth so affected begin to have occasional attacks of soreness. Usually this is not of much consequence and passes away in a few days. This occurs at irregular intervals and grows worse as the disease progresses. Toward the last the paroxysms of soreness become more frequent and are permanently relieved only by the loss of the tooth. When we realize that these periods of soreness occur with tooth after tooth over long periods of time, before the last of them are gone, we must appreciate that this disease has, on the whole, caused a large measure of physical suffering.

Teeth become loose and are finally lost. As the alveolar process is destroyed the teeth begin to have much motion in the remaining part of their alveoli. This loosening may occur when the bony alveoli are but little more than half destroyed. This is effected by the absorption of that part of the bone next to the peridental membrane, and the lengthening and softening of the fibers connecting the teeth with the bone. The fibers are no longer stretched tightly between the cementum and the portion of bony alveolar walls that are left. The teeth then become very loose and may easily be moved about; yet in an attempt to extract them, they are very firmly held by the elongated fibers of the peridental membrane, as by so many small but strong ropes, and resist actual removal. Finally, however, the remaining attachment is so slight that at some time, when a very loose tooth is particularly sore and troublesome, the person will succeed in picking it out with his fingers. In this long and tedious

way, running from five to thirty years, the teeth are loosened and one by one are lost, until finally the person is toothless.

Menace to general health. But this is not all. The mass of decomposing pus and food debris in and about the deposits of calculus, and in the suppurating areas, have continuously been the home of masses of growing bacteria of many kinds, saprophytic and pathogenic, which are often a serious menace to the general health. This may be the most serious phase of the condition, although it has generally received little consideration, either by patient, dentist or physician. The relation of mouth infections to general systemic conditions will be considered under a separate heading.

Variations in the position and progress of the deposit.

I have described above the picture of the injury resulting from the deposit of salivary calculus when it runs its course without interference, attacking all of the teeth together, or in fairly close succession. This occurs only in the minority of cases, which have no treatment. While the deposit on the lower incisors and cuspids is seen most frequently on the lingual surfaces, a beginning may be made upon the labial surfaces also. (See Figures 144 to 147.) Often it occurs that the progress is made mostly upon the lingual and labial surfaces, leaving for a time the septal tissue standing between the teeth almost untouched. Finally the calculus may close in upon the lateral sides of these septi and this tissue will be destroyed. (See Figures 148, 149 and 150.) In the biscupid and molar region, while the principal deposit is on the buccal surfaces, the lingual may become involved, and subsequently the septal gingivæ may be destroyed. The lower bicuspids and molars are usually not quite so extensively involved as the corresponding upper teeth.

Deposit usually confined to certain teeth. Very generally the deposit will be confined to, or a greater amount of deposit will occur on, some certain teeth. Others will escape for a time, or permanently. Then teeth will be lost from this cause only in special regions. These are most likely to be the molars, upper and lower, and the lower incisors, sometimes the one and sometimes the other, or both together. Again, some particular tooth, or teeth, other than the groups named, may be attacked.

conditions contributing to occurrence of deposit.

1. Calco-globulin must be brought to the mouth by the saliva from the salivary glands.

2. Deposits usually occur first on teeth near the opening of the ducts from the parotid or the submaxillary and sublingual glands. Taking all cases together, these are the places where the general bulk of the calculus is deposited.

3. Points of depression in the gingivæ of certain regions or about certain teeth, the thickening of the crests of the gingivæ from any cause, such as mechanical injuries, previous injuries by calculus, etc., may become places of deposit because of the malform.

Form which gives opportunity for initial deposit. The controlling factor then is form which gives opportunity. If we suppose that calculus enters the mouth with the saliva and is immediately ready for deposit, the nearest teeth would receive it. This is the general rule, as has been stated. But the teeth of this locality, and their gingivæ, may be of excellent form, and will not readily receive the deposit, or the soft deposit may be removed in the act of chewing food at the next meal. Then deposits may or may not occur elsewhere, depending upon form which will favor a lodgment.

The position of the deposit is determined by some peculiarity of form which usually will, on close study, be found to furnish a place for the initial deposit and shelter it from removal during mastication. This may be an irregularity in the shape or the position of the teeth attacked, or of their gingivæ. To produce such a result the deviation from normal form need not be great, but just a slight depression of the crest of the free gingivæ and a thickening of its margin, which will furnish a favorable place for sheltering the deposit.

I have had occasion to study this matter very closely in forming traps for the collection of deposits for microscopic study. These traps, as has been mentioned, consist of little gold frames fastened with screws to hold microscopical cover-glasses in selected positions on a plate (See Figure 122.) Once I made a very nice trap for a new plate, beveling the angles and carefully polishing them down to the glass, obliterating the angle of meeting of the gold and the glass, as nearly as possible. No deposit occurred on the cover-glass, although the plate was worn during a number of paroxysms of deposit. I was compelled to restore the angle in order to get a deposit on the glass. An angle or corner, or roughening of the margins of the gold frame, is necessary to give the opportunity for the initial deposit. With such a place of beginning, the deposit will build out over the glass.

Forms of artificial dentures to avoid deposits. This condition should cause us to make a study of the forms of artificial dentures with respect to deposits of calculus. It is quite possible to so make and finish a plate that no calculus will adhere to it anywhere. This requires, first, that all irregularities of surface be avoided; and second, that every part of the plate be finely polished. Every part must be given as nearly a regular surface as possible. All of the embrasures between the curves of the teeth, as these spread from the contact points, should be filled with gingivæ practically as full as these are in the best natural forms. The crests of the gingivæ should be reduced to a fine knife edge where the rubber laps onto the teeth. This will make a surface of rubber or of gold so smooth that it will not receive agglutinin and, as a consequence, no calculus will be deposited upon it. To keep it so in general usage, is of course another question, but it may be done by frequent repolishing. A full upper denture is shown in Figure 168, and a partial denture in Figure 169, both of which have very large deposits.

Influence of mastication in preventing deposits. Occasionally, the influence of the chewing of food in preventing lodgments is strongly accentuated in cases in which an exposed pulp causes pain in chewing food upon one side. This leads the person to do all of the work of mastication upon the teeth of the opposite side. Then the teeth of the unused side will receive the deposits of calculus. In such cases I have seen the teeth of the unused side thickly encrusted with calculus on all surfaces, except some portions of the occlusal surfaces which made contact with occluding teeth, while the teeth of the used side received very little or no deposit, and their investing tissues presented a healthy appearance.

A similar condition is occasionally seen on artificial dentures. If one side is more convenient to use than the other, the unused side will receive and hold deposits, while these will be prevented from accumulating on the used side. Sometimes the upper molar teeth are strongly inclined buccally, and the buccal margins stand out over the buccal surface of the lower molars. This prevents the rubbing of the buccal surfaces of the upper molars by food in the act of chewing, and makes these surfaces favorable places for lodgments.

TREATMENT OF GINGIVITIS AND PERICEMENTITIS CAUSED BY DEPOSITS OF SALIVARY CALCULUS.

ILLUSTRATIONS: FIGURES 171-178.

The treatment of inflammations caused by deposits of salivary calculus should consist: first, of the thorough removal of the deposits and the care of the tissues by the dentist until the inflammation has subsided; second, the training of the patient in the means of preventing a redeposit, gradually leaving to the patient the principal care of the case; third, subsequent examinations at stated intervals to criticize the care by the patient and remove any deposits which may have occurred. It has been sufficiently demonstrated that this plan of treatment is dependable.

The removal of salivary calculus seems to have been regarded as a thankless and disagreeable operation from far back in the history of dentistry. This has been from two causes. First, dentists have had no confidence in the real efficacy of the operation for more than a very temporary benefit; second, they have not understood the nature of the deposit, and while they have recommended to their patients that they keep it off their teeth, they have generally done so in a way which showed an attitude of uncertainty as to results. There has generally been no instruction as to how or when to clean the teeth, further than to direct that the brush be used. The movements of the brush in cleaning have only recently received anything like standardization. Under these conditions it is not much wonder that patients have not succeeded in preventing deposits.

With the discoveries which my own experimental observations of the last few years have disclosed, all of this should be changed. Indeed, previous to undertaking my recent investigations, so far as I had seen results from the persistent plans of treatment of such cases by cleaning operations pure and simple, it had become fixed in my mind that the successful treatment of inflammations of the gingivæ, caused by deposits of salivary calculus, offered but one real difficulty. That difficulty was to convince the patient that a permanent cure could be made by the

cleaning method, and the calculus prevented from lodgment again in harmful quantities. What is now known of the nature of the deposit fully warrants the statement that this may be realized, if the patient will exercise reasonable diligence in the daily cleaning of the mouth in the manner which I will describe. Under these conditions both the dentist and the patient may undertake this work with the feeling that it is well worth the time and the energy put into it.

Removal of deposits and care of tissues by the dentist.

The instrumental removal of the ordinary deposits of hardened salivary calculus, when taken in time, really offers very few difficulties. The deposit is always in sight; if not directly, it may be seen indirectly with the aid of the mouth-mirror. It is practically never buried under the soft tissues, nor covered up. While this statement is true, I have seen a very few cases in which some salivary calculus extended under the free gingivæ. I have also seen the gingivæ when much inflamed and swollen, turned outward from the tooth, creating an open pocket which might become filled with calculus. Such cases are much too rare to enter into any calculation for general cleaning processes. However, their possible existence should not be overlooked.

It should be understood that deep pockets along the sides of the roots of teeth do not occur as a result of deposits of salivary calculus, and they are not therefore to be considered under this heading.

Previous to the removal of the deposits, and as a part of the examination of the mouth, a careful record should be made of each surface of each tooth upon which a deposit is found. A simple and exact method of doing this will be presented later. This record should be the foundation upon which the future conduct of the case should rest. A definite record of the condition of the mouth as to deposits should be the guide for the after care, the training required by the patient and the frequency of subsequent examinations. It is of the greatest importance that the patient shall in the beginning be impressed, not only with the serious final results of neglect, but with the fact that there is a definite and dependable system of handling such cases.

Instruments and instrumentation. There is no more simple operation in the dentist's field than the removal of deposits of salivary calculus. Since the deposit is practically always upon the exposed surfaces of the teeth it is easily seen

either by direct vision or with a mouth-mirror. A very simple set of scalers is sufficient. Years ago these instruments were generally much larger and stronger than those of to-day, because of the frequent necessity of removing heavy deposits. It was not uncommon for the dentist to use a large chisel, held with the edge against the deposit, while the assistant struck it a sharp blow with the mallet. As our people have learned to better control the deposit by brushing, the sizes of the scalers used for its removal have become gradually smaller.

I present herewith a set of six scalers. A similar set may be secured from any dealer in dental instruments. (See Figure 171.) These consist of one pair of pull instruments, one pair of push instruments, a sickle and a cleoid. These are all that are necessary for the removal of the bulk of the deposits. Their use should be followed by selected instruments from the set of scalers for the removal of serumal deposits, which are smaller and are better for removing finer particles which may have been left by the larger instruments. These are shown in Figure 186. In cases in which the deposit is light, the smaller scalers will often be preferred to the larger instruments.

The pull scalers should usually be used first for removing the bulk of the deposit from the lingual surfaces of the lower front teeth. These may be followed by the cleoid or sickle, or both, to remove deposits about the angles or on the proximal surfaces. Oftentimes, heavy deposits on the lingual of the lower incisors, particularly if they extend around the proximal angles, may be removed very nicely by using the pair of push scalers, the blade being applied with its edge part way around the angle into the embrasure.

For the molars, both upper and lower, deposits of salivary calculus may be removed from most buccal, lingual and distal surfaces with the pull scalers. The cleoid or sickle may also be used about the distal angles of these teeth. The push scalers will often be more convenient for proximal surfaces, the blade being used through the space from buccal to lingual.

This operation involves sufficient practice in the adaptation of the instruments to gain confidence and reasonable skill in management. Both the manner of handling the instrument, and the character of the resistance of the material to be removed, must be learned by actual observation and experience. One should aim at the first movement, to place the edge of the pull instrument between the gum and the gingival margin of the calculus, and pull toward the occlusal of the tooth, until force

enough is applied to break the calculus away. If at one time too much is caught to be broken away with the use of reasonable force, the position of the instrument should be changed so as to remove a smaller portion at first. In using the push instrument, care should be exercised to have a sufficiently good finger rest so that the instrument may be prevented from plunging forward with the sudden breaking away of the deposit and injuring the near-by gum or other soft tissue.

In connection with the scaling operation, the dentist or his assistant should frequently flood the mouth with a jet of warm water from a large rubber bulb syringe. This not only keeps the field of operation clear, but is very pleasant and comforting to the inflamed tissues. A water-tank for this especial purpose is shown in Figure 172. This is equipped with an electric thermostat (Figures 173, 174 and 175), which keeps the temperature of the water a little above the body temperature. The style of syringe which I prefer is shown in Figure 176. Great care as to wounding the soft tissues will do much to prevent flooding the area with blood. If much inflamed, the gums bleed very freely. In such cases, the bulk of the deposits should be removed at the first sitting, and the patient should be dismissed for a day or two. At the next sitting, the inflammation should be much reduced, and a more thorough operation may be performed. In such cases in which there is much calculus widely scattered among the teeth, the operation is tedious. Both the patient and the operator may tire out. In such a case the operation may be adjourned from time to time until completed. One should not be in a hurry to get through.

Following the removal of the deposit, at the same sitting, or at a subsequent sitting, if there is much inflammation of the adjacent soft tissue, the surfaces from which the deposits have been removed should be carefully polished with powdered pumice and water, using rubber or wooden disks, or points of various shapes, also orange-wood sticks in the hand. These should all be used with great care not to injure the gingivæ. In some positions, and particularly in cases in which the interproximal tissue has been more or less destroyed, polishing tapes may be used. It should be remembered that the normal attachment of the gingivæ on the proximal surfaces of the incisors is very much closer to the incisal edge than on lingual or labial surfaces, and there is danger of cutting away the proximal attachment in the careless use of strips. In any position, strips should be used with the greatest care.

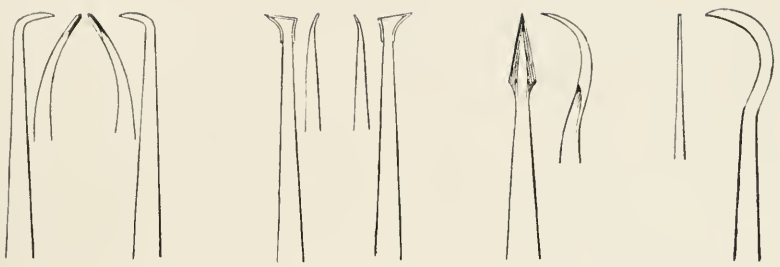

Fig. 171.

Fig. 171. A set of six scalers for removing deposits of salivary calculus. This set consists of two pull instruments, two push instruments, a cleoid form, and a sickle form. If the deposits are slight, the set of instruments shown in Figure 186 will be preferable.

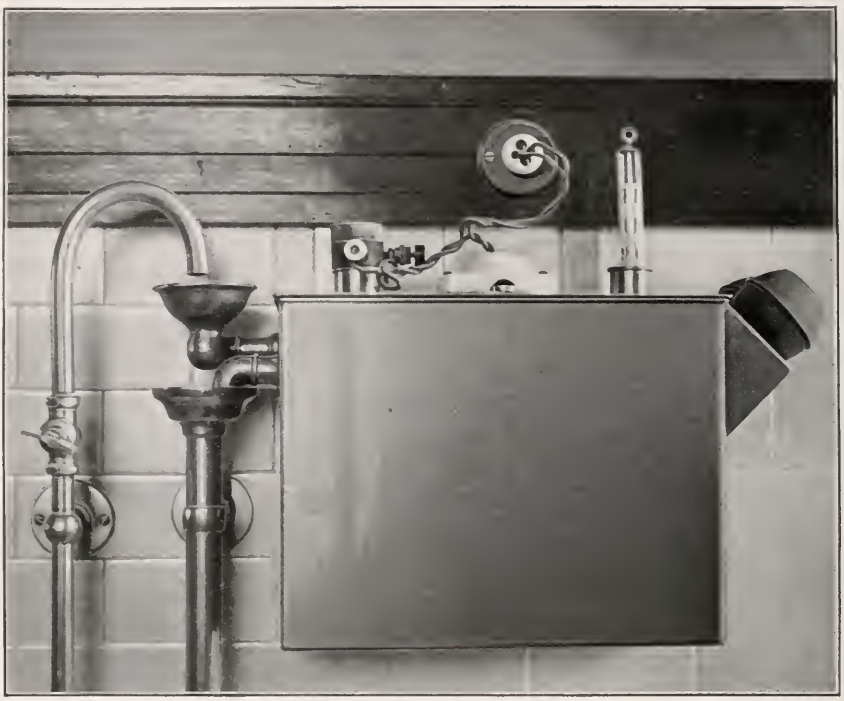

FIG. 172.

FIG. 172. Water tank for dentist's office. This tank is kept full of filtered water and the temperature is regulated by an electric thermostat. The faucet to the left is in a pipe leading from the filter. The valve may be adjusted so that the water drips slowly. The level in the tank is regulated by the overflow pipe which empties into a waste pipe. Heat is furnished by a 16 c. p. carbon filament lamp attached to the porcelain in the center on top of the tank. The lamp is enclosed in a thin copper well which is fastened to the lid of the tank, so that the well is dry. A little red bull's-eye, just in front of the porcelain lamp support, shows when the lamp is on and off. The thermostat projecting from the top of the tank near the left end is described in Figures 173, 174, 175. A thermometer to the right has its bulb in the water. Two rubber bulb syringes fit into "cups" so that the ends of the nozzles are in the water. The tank is not "connected" with the plumbing, and may be lifted off two wall brackets for cleaning.

The water is kept at 103° in the tank, so that it will be about body temperature when the water reaches the mouth. It is very comforting to the soft tissues to flush the mouth frequently during sealing operations, and during cavity preparations as well. The temperature may be kept so close to the body temperature that there will be no pain when washing out the most sensitive cavities.

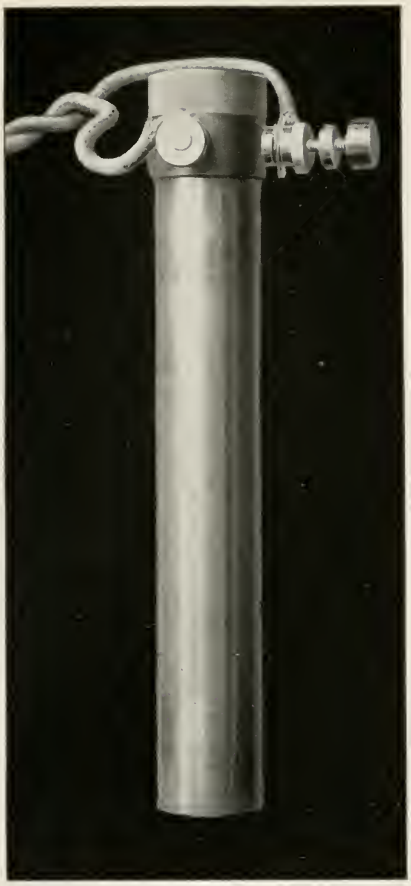

FIG. 173.

FIG. 175.

FIG. 174.

FIGS. 173, 174, 175. Electric thermostat designed by the author for bacteriological and other dry ovens, and for water tanks, as illustrated in Figure 172.

FIG. 173 shows the instrument enclosed in a brass case so that it may be immersed in water. The wires are attached. The connection of the wires with the lamp is shown in Figure 172.

FIGS. 174 AND 175 show the thermostat. The brass rod holds the lower end stationary. Attached to this rod at its lower end is a double metal ribbon consisting of a thin ribbon of aluminum and one of steel riveted together. The aluminum expands more rapidly than the steel, consequently the ribbon is bent with the concavity on the steel side as the heat increases. A platinum point on the upper end makes and breaks the current with another point, which is adjustable with a thumbscrew. (See Figure 174.) By this arrangement the lamp burns whenever the temperature drops below that for which the thermostat is set and is turned off when it rises above.

FIG. 176.

FIG. 176. The large rubber bulb syringe used for rinsing the mouth and for irrigation for all purposes in the mouth. Illustration actual size. This syringe holds two ounces. As these are received from the supply houses, the hole in the end of the nozzle is too small. Enough should be cut from the end to give an opening about 1.5 mm. inside measurement. With such a syringe the mouth may be quite thoroughly cleansed of mucus, blood and debris in sealing operations. In the preparation of cavities it is important to have plenty of water to remove most of the mucus.

Care of tissues by the dentist. During the time of removing calculus, and in the more severe cases for a brief period afterward, the dentist should look after the cleaning of the teeth himself, for it is fair to presume that a patient who has badly inflamed gums from this cause, has not been in the habit of brushing the teeth properly, and could not be expected to do so until after the inflammation shall have subsided. In fact the use of the toothbrush is contraindicated at this time.

In addition to the removal of the deposits, the treatment up to the time when the inflammation shall have subsided, should consist of the most thorough mechanical cleansing with the least possible irritation. This may be best accomplished by using warm physiological salt solution in a rubber bulb syringe. This will remove all accumulations of micro-organisms, their products and other debris from the surface of the inflamed areas and thus advance the healing process. A half-dozen or more syringefuls of the solution should be forced through the interproximal spaces, under and about the gingivæ, giving especial attention to the inflamed areas. The sense of comfort to the patient as a result of several such treatments will often be sufficient to induce the patient to become an enthusiastic user of the syringe in the subsequent care of the mouth. The preparation of the salt solution is much simplified by the use of sodium chloride tablets, prepared for the purpose. (See Figure 177.)

For reasons which will be fully mentioned later, no antiseptics should be used. It should be recognized that the treatment of this condition, with our present knowledge of it, is purely mechanical. Micro-organisms have no influence in causing the deposit of salivary calculus. They do, however, grow luxuriantly in these deposits, and have the principal part in causing the inflammation and suppuration of the soft tissues. Careful observations, over long periods of time, involving large numbers of cases, show unmistakably that if harbor points and masses of accumulations are removed at stated intervals, the soft tissues will not be seriously injured. It has been well known for years that we can not sterilize the mouth for even an hour, nor in any way prevent micro-organisms from growing there in case of lesions in the mouth tissues. If we will prevent accumulations of growth of micro-organisms and their products upon the tissues, the phagocytes will be active in attacking any organisms which may have entered the tissues. The chemotactic action of antiseptics causes the phagocytes to withdraw; antiseptics are therefore contraindicated.

CARE BY THE PATIENT.

As soon as the inflammation subsides the patient should be trained in doing the necessary cleaning. This is just as important as the removal of calculus. The dentist should avail himself of every opportunity to impress this fact. Indeed this is the large part, without which the operation can be of only temporary benefit. There is nothing permanent in the simple removal of calculus. Permanence must depend upon the daily habit of the patient. It must become a part of the patient's care of his or her person. Every one should remember that distinctly. It is the dentist's duty to do this teaching, and in no such case to discharge the patient as well. Do what one will, with our present knowledge of this subject, the tendency will remain for the deposit to recur. We may train certain patients to be so careful in their eating that no deposits will occur and at the same time do much to improve the patient's general health and vigor. That, however, is not fully dependable. It is much easier for patients to control the matter by cleaning than by care in eating, and this is, therefore, the safer treatment. As a matter of fact both should go together. We know full well that soft calculus remaining on the teeth only a few hours does no appreciable harm.

In many of these cases it is difficult to say at what point the dentist should transfer the care of the case to the patient. In all cases in which there is any marked inflammation of the investing tissues, the case should, as already mentioned, be kept under observation until the inflammation has subsided sufficiently to permit the free use of the brush and syringe by the patient. In the majority of cases, even those in which there is much inflammation, the improvement is rapid. Within a few days the patient should usually undertake the cleaning operations. Then the visits to the dentist may be at greater intervals.

Some patients will be very willing to do their part, but be very awkward at first, and will need watchful care and instruction. Others will take it up easily from the start. The most difficult thing is to instil the idea of perfect regularity in doing this cleaning. Even with willing patients, it is difficult to accomplish this, although it is absolutely essential to success.

As a first step in the training of the patient, the dentist should point out the places where the deposit has occurred and explain how necessary it is that these areas be brushed at least twice daily, if redeposits are to be prevented. The patient should understand that the deposit is soft at first and remains so

for twelve hours or more, and that during this time it may be easily removed with a brush and plain water. Absolutely nothing else is required. Particular emphasis should be laid on the fact that to miss the brushing of these areas a single time may mean that there will be sufficient hardening of a slight amount so that it can not be removed with the brush, also that this slight deposit, by its roughness, serves to attract and hold future deposits.

The dentist should see to it that the patient has proper brushes, and a syringe, and he should by using his brush in his own mouth demonstrate the positions and movements necessary. He should, in many cases, require patients to bring their brushes to the office, observe their use of them, and instruct them in the proper methods.

The use of the rubber bulb syringe by the patient as a part of the daily routine of cleaning the mouth will be appreciated by most patients when they have once learned its use and the sense of comfort which it gives. While deposits of salivary calculus may be prevented with nothing else than the brush and water, the gingivæ may be kept in better condition by the use of the syringe and thereby reduce the opportunity for future deposits.

A detailed statement of the technic of using tooth-brushes and the syringe will be presented under the heading of Mouth Hygiene.

From what has been said, it should be quite obvious that tooth-powders, tooth-pastes and mouth washes are not indicated in the treatment of these inflammatory conditions.

Subsequent examinations.

Every patient for whom deposits have been removed should be impressed with the importance of returning at stated intervals for inspection and the correction of errors in cleaning. The frequency of such visits should depend upon the case, and the earnestness of the effort on the part of the patient. In cases in which there has been serious neglect, the patient should be requested to return within a month. If it is then apparent that the cleaning is being well done, a longer period may be given before the next visit. For persons who have become well trained and are in earnest in the care of their mouths, appointments every six months are sufficiently frequent, and for many such people, little or no deposit will be found even then.

As will be presented more in detail later, the dentist should

have a reliable plan of arranging for subsequent examinations. The patient should be informed of the desirability of an examination at a stated time, and the dentist should offer to take the responsibility of notifying the patient when the time arrives. This must be done on a plan by which there is practically no danger of failure in the sending of such a notice, for if the dentist assumes the responsibility of notifying the patient, and then fails to do so, the patient will have good cause to blame him. Many patients will welcome such a plan and will be so impressed with the whole scheme of treatment and the interest manifested by the dentist, that they will undertake their part more earnestly.

On the occasion of each subsequent examination, the dentist should refer to his previous examination record, and should make a careful inspection of all positions from which deposits were removed to note how successful the patient has been in the cleaning. Whenever a deposit is found, it should be pointed out to the patient, and directions given for the better care required in the future. At the same time a record should be made of whatever deposits are found and the patient should know that this is done. Then the deposit should be removed.

Such a plan of recording places of deposit, and checking up the care by the patient at each sitting, together with the education of the patient as to the nature of the deposit and training in the cleaning necessary to its prevention, all carried out with an enthusiasm and earnestness on the part of the dentist, will not fail to procure the earnest coöperation of most patients. This is especially true if the patient's attention is called to the almost certain eventual loss of those teeth which are neglected. For such persons this plan of treatment will not fail. It is dependable.

Patients who can not be induced to take at least fair care of their mouths, may as well be advised that their cases are hopeless.

Fixation of teeth that have been loosened as a result of deposits of salivary calculus.

It not infrequently happens that teeth which are loosened as a result of the destruction of a part of their investing tissue by the deposit of salivary calculus will with proper treatment again become tight. If, following the removal of the deposit, the teeth are kept clean, the remaining tissue of the peridental membrane may shrink down and hold the teeth firm

FIG. 177.

FIG. 177. Bottle containing 100 sodium chloride tablets and an ordinary drinking class (illustration actual size) containing eight ounces of water. Two salt tablets of 16⅖ grains each should be added to eight ounces of water to make a physiological salt solution. It is recommended that patients prepare the salt solution in this way for home use.

Fig. 178.

Fig. 178. Radiograph of a case in which an appliance is holding the four lower incisors together, although the centrals have lost almost all of their bony attachment.

against direct pressure in mastication, if the occlusion is squarely down upon the teeth. If, however, the teeth should be inclined, or the wear of the surface should be more on the buccal than on the lingual, or the reverse, the force exerted is inclined to drive the tooth to the one side or to the other. In cases in which the destruction of the membrane has not been too great, the loose teeth may be fixed in position by an artificial appliance which will hold them against lateral strain, and thus give the remaining investing tissues opportunity to regain their normal tone. Many of these shaky teeth will, as a result of the reduction of the swelling around the apex of the root, the thinning down of the membrane and the apparent strengthening of the fibers, do service for many years, if they are kept clean. After several months the fixture may be removed. After the teeth have become firm, they will usually do better without the appliance, because they can much more readily be cleaned.

Whatever form of appliance is used, it should be so constructed that it will be entirely free from the soft tissues, being attached to the crowns as far away from the gingivæ as possible. It should be so made for the double purpose of avoiding irritation to the gingivæ, and to permit the most thorough cleaning of the portions of the teeth near the gingivæ. Fixing such cases with appliances that reach to the gums makes proper cleaning impossible and invites deposits of calculus and food debris. The danger of such foci in the mouth to the general health has been pointed out, and it is unquestionably better to extract the loose teeth than to retain them under such conditions.

There are many ways in which such teeth can be fixed in position. Comparatively narrow bands may be fitted to the crowns without impinging on the margins of the gingivæ. These bands should include the loose tooth or teeth and at least one firm tooth on either side. In cases in which such an appliance is to be placed on the lower incisors, the bands may be made tolerably heavy and well reinforced with solder, so that the labial portion for each tooth may be cut away after the appliance is cemented to place. This will then show of the gold only what will appear to be a row of proximal gold fillings. Lingual plates may be swaged or cast, and fitted with small platinum pins which extend part way or entirely through the crowns of the teeth, being placed far enough incisally to avoid the pulp chambers.

Other plans will be quite as satisfactory. It is not within the scope of this book to describe these in detail; only to call

attention to the advantage of such appliances in some cases in which the teeth are loose, but have a considerable portion of their investing membranes remaining. When teeth have become so loose, on account of the extensive destruction of the investing tissues, that they will not be likely to again become tight, they had better be extracted at once, and replaced by a bridge or some other appliance.

The rule should be that a tooth which can be used without too much discomfort, should not be held in place with an appliance. The tooth will become fixed in its position, if not driven out by some other forces mentioned above, much better if left without a band. One effect of the motion of the tooth in the chewing of food is a stimulation to the remaining portion of the membrane about the root, which serves to strengthen the fibers and the portion of alveolar process about it, so that these tissues become strong enough to support it. This is to be sought usually, instead of supporting the tooth by bands. Bands give the patient comfort, it is true, for the time being, but generally the fancied security contributes to negligence in the cleaning process and the teeth will be lost sooner than if the bands had not been used. (See Figure 178.)

Some of the best recoveries of teeth which have been loose, under my own observation, have resulted from redoubling the effort of cleanliness and depending upon patients to avoid hurting the teeth until the natural reparation forces have tightened them. I have seen teeth which were very loose, notwithstanding the fact that there was an abundant amount of peridental membrane about the roots to hold them firmly in position, if the inflammation could be controlled. It is only those which are liable to be forced out of position by the pressure of mastication that should have bands for fixation. When the occlusion comes squarely upon the teeth, they will do better without bands.

An interesting case of a very loose tooth which became tight as a result of exceptionally careful cleaning is reported under training in cleaning the mouth in the discussion of Mouth Hygiene.

GINGIVITIS DUE TO DEPOSITS OF SERUMAL CALCULUS

ILLUSTRATIONS: FIGURES 179-185.

This distinct type of gingivitis is caused by the deposit of serumal calculus on the enamel in the subgingival space. The term serumal calculus is applied to the deposit occurring on the enamel under the free margin of the gingivæ, or on the cementum after the peridental membrane is detached. The term was first suggested in a paper* which I read before the Illinois State Dental Society in 1882. The late Dr. L. C. Ingersoll, of Iowa, had used the term *sanguinary calculus,* and contended that it was a deposit from liquor sanguinis that exuded from the tissues in a state of irritation or inflammation. I believed then, as I do now, that the first deposit of this calculus is brought to the subgingival space by the *normal subgingival fluid,* or possibly during some excitation of the normal flow. In the absence of a definite gland this fluid should be termed a serum — an exudation, rather than a secretion.

Causes of deposit and conditions of occurrence.

The underlying causes of the deposit of serumal calculus are not different from the causes of deposits of salivary calculus. As previously mentioned, the subgingival spaces are kept constantly moist by the exudation of a serum from that portion of the free gingivæ which normally lies in contact with the enamel. Under normal physical conditions this serum contains no calco-globulin, but under those conditions already described under which calco-globulin is present in the fluids secreted from the salivary glands, we would also expect to find a calco-globulin present in the other juices and fluids of the body, and excreted with them. The serum which bathes the subgingival spaces should contain its proportion of calco-globulin, and depending on the quantity, it might be expected that this would find a place of deposit on the enamel of the subgingival space. Doubtless much of it passes out from the subgingival space and becomes

* Phagedena Pericementi, by G. V. Black. Proceedings Illinois State Dental Society, 1882, p. 93. See p. 98, second paragraph.

mixed in the saliva, while that which remains is not disturbed by the cleansing of mastication, nor by the artificial cleaning methods ordinarily employed.

Therefore, it seems logical to state that there may occur a more or less general deposit of serumal calculus on the enamel of the subgingival spaces, as a result of the excretion of calco-globulin with the serum which is normally discharged into these spaces. We might also find an excessive deposit of serumal calculus on the enamel of certain subgingival spaces as a result of irritation or inflammation of the overlying soft tissue, because under such conditions an excessive amount of serum, which might be charged with calco-globulin, would be poured out into these spaces. Or we might find a deposit of serumal calculus in a single subgingival space and nowhere else, in a case in which the tissue overlying the particular space was irritated or inflamed, causing an excessive outpouring of serum. The amount of calco-globulin in the serum might be so slight that in all spaces not subjected to irritation, there would be insufficient quantity of calco-globulin brought to the spaces over a long period of time to make an appreciable deposit, while the increased discharge from the irritated tissue might bring a sufficient quantity to give a definite deposit in a comparatively short time.

It should also be recognized that some individuals apparently practically never have an excess of calco-globulin discharged with the saliva or other secretions, and we should not expect to find deposits of serumal calculus in the mouths of such persons, even in those spaces where the inflammation of the overlying tissue is marked. However, since most of the inflammations of the gingivæ are permitted to go on year after year without attention, there will usually be a deposit in such places, because it is probable that few persons are continuously free from paroxysms of excess of calco-globulin.

We should generally expect to find deposits of salivary calculus in mouths in which deposits of serumal calculus are present, as the same condition would result in the outpouring of calco-globulin in the saliva and subgingival fluids simultaneously. The deposits of salivary calculus need not necessarily be present, however, because the local conditions in those positions in which salivary calculus would generally be found, may be unfavorable for an accumulation of the deposit; or any accumulations which have occurred, may have been removed by

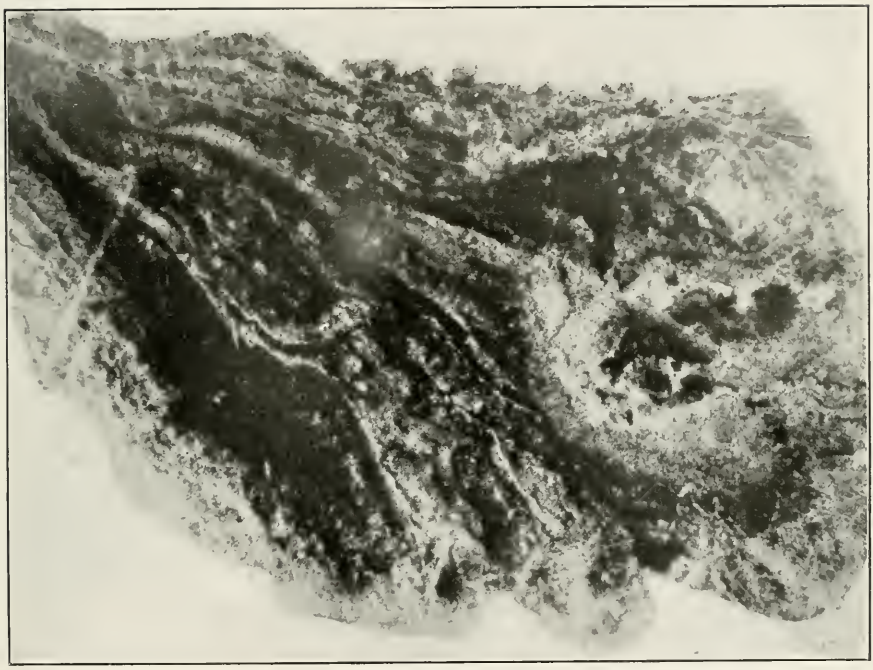

FIG. 179.

FIG. 179. Photomicrograph from a section of a crumb of very black serumal calculus. (See description of process of grinding, in Appendix of this book.) The outer surface is the lower border of the picture, upon which accretion was in progress. It gives a slight showing of masses formed by spherules of globulin. The irregular veining shows lines of accretion.

FIG. 180.

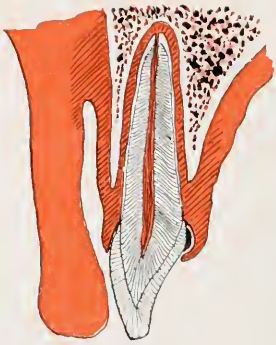

FIG. 181.

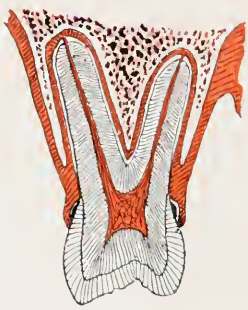

FIG. 182.

FIGS. 180, 181, 182. Drawings to illustrate the positions in which deposits of serumal calculus occur on the surface of the enamel in the subgingival space. Deposits in this position are usually flat scales, while those on the roots are more generally nodular.

FIG. 180 shows a deposit of serumal calculus under the free gingiva on the labial surface of the enamel of a lower incisor tooth.

FIG. 181 shows a similar deposit on the lingual surface of an upper incisor. Suppuration of the peridental membrane, resulting from deposits in this position, causes the teeth to move labially, and such cases are generally hopeless, after much progress has been made. (See Figures 260, 261.)

FIG. 182 shows an upper molar with deposits of serumal calculus on the enamel of both the buccal and lingual surfaces.

the vigorous use of the teeth in mastication, or by artificial cleaning, neither of which would disturb the serumal deposit.

It should generally be the case, however, that no deposits of salivary calculus will be found in those mouths in which well-marked inflammations of the gingivæ have existed for a considerable time, say a year or more, without any deposit of serumal calculus having occurred in such positions. In such cases there would have been a continuous outpouring of an excessive quantity of serum from the inflamed areas, and if there had been insufficient calco-globulin to form deposits in such positions, it would be expected that no calco-globulin had been secreted with the saliva during the period of inflammation.

Comparison of serumal with salivary calculus.

Serumal calculus is more compact and is harder than salivary calculus. Its color is a dark brown or black, often intensely black, and if broken, it is lustrous when washed and dried. No analysis of this calculus seems to have been published. This is probably because of the difficulty of obtaining it in sufficiently large quantities. Formerly, I had supposed that serumal calculus was a different order of deposit from salivary calculus, or that it was a crystalloid* form instead of a granular form such as we find in salivary calculus. Since I have been able to grind fine sections of the two varieties for microscopic examination, I have found little difference in their structure, except that the serumal deposit is more dense. (See Figure 179.)

Gingivitis due to the deposit.

The first effect of a deposit of serumal calculus in the subgingival space is to cause an irritation of the gingivæ. If my supposition expressed above is correct, this irritation tends to an increase in the deposit by causing more serum to be poured into the space, and this in turn increases the irritation. The deposit adheres very closely to the enamel of the subgingival space. (See Figures 180 to 185.) The overlying free gingivæ have a darker cast than normal, caused by the dark color of the calculus showing through the tissues which cover the deposit.

* The term crystalloid indicates a form which is similar to a crystal, but is not a true crystal form in the chemical sense. The crystalloid form, as found in the bodies of animals, always has a basis of colloid material. The calco-spherites are a similar form of deposit. Indeed, there are no crystals in organic matter or material used by life force in constructive metabolism. Many compounds crystallize out from solutions when the life force lets them go. Even the enamel rods, that contain the least organic matter of any organic material, are not crystallized. Enamel does not decompose light, as its principal chemical compound will do when dissolved and crystallized as an inorganic body.

In positions where the gingivæ are thin, the deposit underneath will show through as a bluish spot in the soft tissue. If the deposit is removed the spot disappears.

Compression of deposits by the gingivæ. I have thought the greater density of serumal as compared with salivary calculus might be due to compression from the gingivæ in forming the mass when soft. The form of the usual scale and its smooth, rounded surface would indicate this. It is probable that during any one of the inflammatory periods the grasp of the soft tissue upon the tooth or upon a previous deposit of calculus is relaxed by the swelling of the tissue, giving room for the deposit. Then the soft mass seems to relieve this irritation, and the recovery of the tissue causes it to draw tightly over the soft deposit and smooth it down into the form of the scale which occurs in this position. In this way the mass grows.

Variations in location of deposits.

In many cases the deposit of serumal calculus is confined to small points on the buccal, labial or lingual sides of the teeth, rather than the proximal sides. We will, however, sometimes find rings of this calculus which completely encircle the teeth. (See Figures 183, 184 and 185.)

My personal observation has been that, while none of the teeth are exempt from deposits of serumal calculus, it occurs most frequently in the front part of the mouth, especially on the incisors and cuspids, which give the greater number of cases, and the bicuspids a less number, and the molars the least. The third molars are involved more frequently than either the first or second molars.

It is very liable to involve and destroy the membranes of one or two or several teeth and leave others uninjured. This occurs because of the fact that a suppurating pocket once established will go on and on, often very slowly, but continuously, until the tooth is lost. As has been mentioned, there may be a general deposit in many or all subgingival spaces, or in any particular position in which an irritation of the gingiva has occurred.

Suppuration involving peridental membrane.

The greatest danger from the inflammations caused by these deposits is to the attachment of the peridental membrane.

As the inflammation increases, suppuration usually occurs. The point of the beginning of suppuration is at the gingival

line, and the attachment of the peridental membrane is first destroyed. As this progresses, the gingivæ may shrink away and become shorter, exposing the first deposit. Then another line of deposit, if it has partly encircled the tooth, is apt to occur in the space gained. By this time, the suppuration of the adjacent peridental membrane may form a pocket along the side of the root of the tooth and a suppurative pericementitis has been established.

I remember one case in practice in which I found a little pus pocket confined to the disto-labial angle of an upper lateral incisor, caused by a small nodule of serumal calculus. There were also several small nodules elsewhere in the mouth, but this was the only point of suppuration. This one point healed readily by a little careful handling, but afterward there was a deep scallop in the crest of the free gingiva at that point, because of the detachment of the peridental membrane from the tooth and the injury of some portions of other supporting tissue by suppuration. This patient was frequently in my chair, for ten years afterward, but there was no other deposit of serumal calculus. Neither was there any betterment of the deformity of the gingiva by reattachment over the space of the destruction of the fibers of the membrane.

TREATMENT OF GINGIVITIS DUE TO DEPOSITS OF SERUMAL CALCULUS.

ILLUSTRATIONS: FIGURES 186-188.

The treatment of inflammations of the gingivæ, caused by deposits of serumal calculus, should be along the same general lines as the treatment of inflammations caused by deposits of salivary calculus. The principal points of difference are in the instruments used and technic employed in removing the deposits, and the greater importance of the rubber bulb syringe both in the care by the dentist and in the after treatment by the patient. In those cases in which, in addition to the deposit of serumal calculus, there is apparent an exciting cause of the inflammation, other than the deposit, it will, of course, be necessary to remove such cause, whatever it may be. The different injuries to the gingivæ and their treatment, are considered elsewhere.

The treatment should consist: First, of the thorough removal of the deposits, and the care of the tissues by the dentist until the inflammation has subsided; second, the training of the patient in the means of preventing redeposits; third, subsequent examinations at stated intervals to criticize the care by the patient and to remove any deposits which may have occurred.

Removal of deposits and care of tissues by the dentist.

The removal of deposits of serumal calculus is an entirely different matter from the removal of deposits of salivary calculus. Serumal calculus is deposited in the subgingival spaces, is covered by the free gingivæ, and hidden from view. The deposits are usually in the form of flattened scales which cling very tenaciously to the surface of the enamel. It requires a very sharp instrument and considerable force to dislodge them. Care should be exercised to do the least possible injury to the gingivæ in connection with this operation.

As mentioned in connection with the treatment of gingivitis caused by deposits of salivary calculus, a careful record should be made of the positions in which deposits are found when the mouth is examined. This record should be made before the

FIG. 183. FIG. 184. FIG. 185.

FIGS. 183, 184, 185. Three molar teeth showing "rings" of deposit of serumal calculus on the enamel of the subgingival space. Frequently these "rings" encircle the crown. Specimens from Northwestern University Dental Museum. Figure 184 was presented by Mr. Harold A. Hooper, a student in the school.

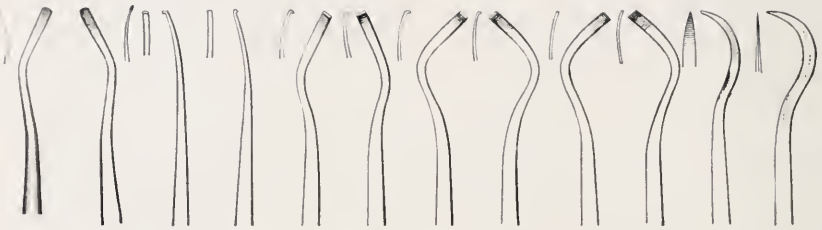

Fig. 186.

Fig. 186. A set of scalers for removing deposits of serumal calculus. These were designed especially for the removal of deposits from pus pockets and the directions for their use are given in discussing the removal of deposits in the palliative treatment of chronic suppurative pericementitis. (See Figures 268 to 282.)

operation, or while the removal of the deposits is in progress, and it should be verified and corrected, making additional entries for any deposits found which were not previously noted. This record should be made with the intention of referring to it and comparing the conditions found on subsequent examinations.

In the removal of serumal calculus, different instruments from those for removing salivary calculus should be used. These should be narrow, flat blades. These blades should have square ends, ground with very sharp edges for use with a push motion, or should be armed on the flat side with a short blade, in hoe form, with a cutting edge, which may be used with a pulling motion. These edges should be kept very sharp to be effective. The deposits may be well removed by either form of instrument. Some men have preferred one of these forms, some the other. In the use of either form, the greatest difficulty is to succeed in removing the calculus without injuring the attachment of the peridental membrane at the gingival line.

In the actual operation, the calculus is found and its position clearly mapped out by the movement of the sharp edge of the blade over the enamel of the subgingival space. One of the first things the student should learn is to detect serumal calculus by the sense of touch. This he will do best by passing any one or several of the instrument forms over serumal deposits which have been discovered, and noting carefully the sensations conveyed to the fingers by the motion of the instrument end over the calculus. One may soon learn to determine very accurately the form of nodules, or even of very thin scales, in this way.

It will be found that the form of the deposit will vary from small nodules to very thin scales, the moderately thin scales predominating. Many of these will be actually in contact with the attachment of the soft tissue at the gingival line. Suppuration will have caused the detachment of more or less of the adjacent peridental membrane about others. Some will have been deposited a little apart from the attachment of the soft tissue at the gingival line.

By referring to the accompanying illustration (Figure 186), it will be noted that there are twelve instruments in the set of scalers designed for the removal of these deposits. These consist of three pairs of pull scalers, and one pair of push scalers, one instrument of sickle form and one cleoid or claw form. The set also includes a pair of peridental membrane explorers. These have smoothly rounded ends and were especially designed to examine the line of attachment of the peridental membrane.

In the use of the pull form of the instrument the edge of the blade should be slid under the free gingiva onto the calculus, and over it until the blade is felt to drop against the enamel beyond the margin of the deposit. Then pressure and a sharp pull should be made in the endeavor to bring the scale or nodule away. Care should be exercised in the manipulation of the instrument not to pass it too far beyond the deposit and thus injure the tissue attached to the root. On the other hand, if the edge of the instrument is not passed over the margin of the deposit, the instrument may slip over it and bring nothing away. It requires considerable practice in this instrumentation to do it deftly. All of the sensations of sliding the instrument to contact with the deposit, of being lifted onto it, of dropping over its edge and coming in contact with the attachment of the soft tissue at the gingival line should be carefully studied. The whole matter becomes easy of accomplishment as one acquires dexterity.

The motions of the push scalers are the reverse of those of the pull scalers. The edge of the blade is lodged against the margin of the scale, and a push made to dislodge the deposit. In using these instruments, there should be a secure finger rest, so that the danger of the instrument plunging ahead into the soft tissues will be avoided.

One of the difficulties occurs when a thin scale breaks up, instead of coming away. Then individual broken parts must be searched out and removed, until nothing more can be found.

The operator should usually see the patient again within a few days, and examine very carefully for particles which were overlooked at the first sitting. These may be shown by points of redness of the gingivæ, or may have to be searched out with the explorer. In a large proportion of cases the explorer may be introduced, and in cases of doubt, the free gingivæ may be lifted a little apart from the tooth, so that one can see into the subgingival space for the detection of very thin scales. In doing this great care should be exercised not to injure the gingivæ and cause them to stand off from the teeth. In cases in which a number of teeth have deposits, the operation is apt to become very tiresome. The treatment should not be done hurriedly, but should be adjourned to another sitting whenever one becomes fidgety over the search for small particles. All through this operation the syringe and warm water should be at hand for washing the subgingival spaces for the removal of broken particles of deposit and clearing away blood which may impede the operation.

Care of the tissue by the dentist. The after treatment of the gingivæ, following the removal of serumal calculus, is simple. They should have a very thorough washing from the syringe loaded with physiological salt solution, using it plentifully, and in such manner as to stretch the free gingivæ open and wash the subgingival spaces clean. This should be carefully explained to the patient, and, during the washing, the patient should be asked to note particularly the sensation of the stretching open of the subgingival spaces by the stream of water, so that he or she may know when they have this sensation in their own efforts at subgingival cleaning. No other treatment is necessary. No medication is indicated. In the cases now under consideration, in which the inflammation is confined to the gingivæ, and there has been little or no detachment of the peridental membrane, the tissues will return to their normal condition within a few days.

Care by the patient.

The most important training which the patient should receive for the after treatment of this condition, is in the use of the rubber bulb syringe. (See Figures 187 and 188.) If redeposits are to be prevented, this must be accomplished by cleansing the subgingival spaces so thoroughly and so frequently that all of the calco-globulin which is brought to the spaces will be removed before it becomes hard. Yet this is probably not the most important function of the syringe, for by its use the gingivæ are maintained in better health and this greater healthfulness prevents the disposition to the deposit of serumal calculus. Such cleaning must be done without irritation to the gingivæ. This may be easily accomplished by the patient by washing these spaces twice daily with the rubber bulb syringe and water. This plan has been followed for several years by a sufficient number of patients to prove its efficacy.

The greatest difficulty will be met with in the matter of impressing each patient with the importance of this twice daily cleansing with the syringe. The patient may use plain water or physiological salt solution. The latter will be preferred by many. As previously mentioned, they should be taught to recognize the sensation as the water lifts the gingivæ away from the enamel in space after space as the syringe is passed around the arch. Many persons will be quick to appreciate the added comfort of the mouth after each washing, and will promptly become enthusiastic in the use of the syringe. Others will do little or nothing with it. Just in proportion as the dentist is

able to impress his patients with the serious final results of the inflammations caused by the serumal deposits, and the very important part which their own care must play in prevention, will he succeed in securing their coöperation. The technic of using the syringe will be presented under Mouth Hygiene.

No other treatment than this washing is necessary, except it be the massage with the tooth-brush in the usual cleaning methods. Certainly nothing is to be gained by the use of mouth washes, tooth-powders, etc., in the care of this condition. It is entirely a question of the thorough mechanical cleansing of the spaces.

Subsequent examinations.

When each patient is dismissed, there should be an understanding regarding the time when the next examination should be made. If it is agreed that the dentist shall notify the patient when the time arrives, a memorandum should be made to carry out such an arrangement. How soon the patient should return will depend entirely upon conditions. It may be desirable to set a time within a month, if for no other purpose than to make some inquiry as to how the cleaning with the syringe is progressing. For most patients who have become skilled in the use of the syringe, an examination every six months will be often enough.

The record of the previous examination should be consulted, and every place where a deposit was found previously, should have especially careful inspection. Points at which there had been some suppuration of the tissues should be seen frequently, on account of the greater danger of a recurrence. Such a suppuration, unless very shallow, is likely to cause a slight depression of the gingivæ, with a corresponding deviation in the even curve of the crest. A new record should be made at each examination, so that the full history of each case will be filed for reference. In time these case histories will become of great value.

Fig. 187.

Fig. 187. Rubber bulb syringe for patients to use. Illustration actual size. The bulb holds 1½ ounces. This is a more convenient form for patients than the one shown in Figure 176. The hole in the end of the nozzle should be the same, 1½ mm. About ¾ of an inch must be cut off the nozzle as ordinarily supplied, in order to have a sufficiently large hole. The use of this syringe is shown in Figure 188.

FIG. 188.

FIG. 188. The position of the rubber bulb syringe in washing the subgingival spaces. The end of the nozzle should touch the enamel of the tooth near the crest of the gingivæ as it is passed along the arch, the angle being such that the water or solution will be forced into the subgingival spaces. This is the most effective means of preventing deposits of serumal calculus in the subgingival spaces.

GINGIVITIS CAUSED BY INJURIES

ILLUSTRATIONS: FIGURES 189-215.

The gingivæ stand in the position of protection to the deeper investments of the teeth and their attachment in their bony alveoli. These attachments, and the preservation of them, are of first importance to the functions of the teeth. It is our duty as dentists to look as closely after the health of these tissues as that of the hard tooth tissues, and to conserve and protect them from injury to the fullest extent possible.

We have already reviewed the injuries which occur as a result of deposits upon the teeth from the secretions which are poured into the mouth from its secretory glands. We have noted the inflammations caused by deposits of salivary calculus, resulting in the gradual destruction of all of the investing tissues of the teeth progressively toward the apices of the roots; also the gingivitis caused by deposits of serumal calculus in the subgingival spaces. Later we will consider the progressive destruction of the attachment of the peridental membrane resulting from such a gingivitis.

Formerly it was generally believed that most of the cases of destructive diseases of the investing tissues of the teeth were caused by deposits of calculus, and unquestionably the percentage of cases due to deposits was much greater in former years than now, the gradual reduction being due to the better care of their mouths by our people. A critical examination of the mouths of a large number of adults will establish the fact that the majority of cases at the present time result from slight traumatisms and irritations of the gingivæ, and that deposits of calculus are the first cause of a minority. Most cases are later complicated by deposits, and these have often been mistaken for the exciting cause. It frequently requires very careful study of cases, especially those which have made considerable progress, to determine the beginning or first cause.

There is no more promising field for the study and practice of prevention than in the group of conditions which are the exciting causes of the inflammations of the gingivæ. Most dentists seem not to have recognized many of these causes at all;

or if they have recognized them, they have failed to appreciate the direct relationship between the apparently trivial gingivitis and the more serious lesion of the peridental membrane which results. It should be understood that a gingivitis precedes the pericementitis in every case, and in view of what has been said relative to the lack of power of reattachment of the peridental membrane to the cementum, it is of the utmost importance that we pay more attention to the earlier lesion, the gingivitis, which can usually be prevented or cured by very simple means.

General Statement of Causes and Symptoms.

Faulty contact points* constitute the chief factor in the causation of this form of disease beginning in the bicuspid and molar region, where the heavy work of mastication is done. Within my observation more cases have been caused by faulty contacts than any other forms of injury.

If, for any reason, there is a slight opening between two teeth, food which is tough and stringy will be crowded into the interproximal space and cause pressure upon the septal tissue. Or, if the contact is too broad, stringy foods, such as fibers of beef or chicken, are likely to be caught between the flat surfaces and held there, thus injuring the septal tissue. A single such occurrence does but little harm, provided the food is promptly removed, but this having occurred a few times, is liable to occur frequently, or even at each meal at which meats or other stringy foods are eaten. This frequent impaction of food against the septal tissues finally becomes habitual, and the tissue is more and more injured by compression.

Inflammation. As a result of this repeated pressure and irritation the tissue is inflamed much of the time. In the earlier stages there may be redness of the gingiva in the particular space, and the festoons will be slightly swollen. The impaction of the food against the central portion of the septum may press the buccal and lingual portions outward in their respective embrasures. The tissue will show marked redness at intervals, and lapse into a sluggish condition of chronicity between times,

* In speaking of the contact made by the contact points between two teeth as they stand in the arch, I have used certain abridged phrases. For instance, the touching of the contact points upon each of two teeth is spoken of as *the contact*, or *the contact point;* as, *the contact* between the first and second molars, or between any other teeth; or, *the contact point* was rough, or *the contact* was weak, etc. The expressions seem to have an aptness which recommends them, and I have therefore made no effort to eliminate them from my writing.

when many observers would be inclined to regard the appearance as healthy.

Suppuration. After a time suppuration occurs in the secluded space between the teeth, affecting especially the attachment of the peridental membrane to the cementum. There has been so little opportunity, with the ordinary methods of cleaning, to prevent the growth of micro-organisms in these places, with the continuous reinfection which they afford, that a chronic suppuration is maintained. This may be of such slow progress as to require years to accomplish the destruction of the peridental membrane to the point of loosening of a tooth, or it may proceed more rapidly.

Complaint of pain variable. There may be complaint of pain during the time food is crowded against the septal tissue. Some patients will complain that food has been getting between certain teeth, and they are unable to continue a meal until the impacted food has been removed. In the examination of the mouth of another patient, the dentist may find one, two, or several septal gingivæ threatened with serious injury from lodgments of food; or possibly the case will have already progressed so far that the eventual loss of the teeth is inevitable; or there may even be considerable amounts of food debris between the teeth at the time of examination, and yet the patient will insist that no inconvenience has been felt.

I remember well a case in point, occurring in the mouth of a man of prominence in his community. He made an engagement to see me regarding extensive abrasion of his teeth. When he sat in my chair I at once saw that the septal gingivæ between the upper first and second molars on both sides were practically destroyed, and those between many other teeth were seriously injured. He said emphatically that he had had no pain or inconvenience, and that he could not conceive that serious disease existed in his mouth.

I made an engagement with this man to take lunch with me the next day, without disclosing my object, which was to see him chew food on the lame teeth. To my surprise he ignored the condition absolutely and chewed broiled beefsteak as if the teeth were well. I refused to build up his worn teeth with gold, with the statement that I could not do it in such a way as to benefit him. The fact was I was unwilling to make such operations in teeth which to all appearances would very soon lose their membranes. Within the next year the case had progressed to the point when certain teeth were loose and occasionally sufficiently

sore to cause much discomfort. This man died some years later, as I now believe, from infection originating in his mouth. He was reported as having died from a recently developed rheumatic condition in which heart symptoms occurred.

In this class of cases the dentist must be prepared for indifference by patients who are even in serious condition; and also to hear great complaint in cases found to be trivial, or even in those of which one can find no sign of inflammation and no apparent cause at a first examination.

This much as a general statement regarding the condition of pain. It is utterly unreliable as indicating the gravity of the condition. Our treatment of these cases should be based entirely on the conditions presenting, without regard to complaint or lack of complaint by the patient.

Absorption of septal tissue. As time passes, some absorption of the septal tissue will occur, beginning in the central portion bucco-lingually. The absorption will progress gradually until the septal tissue may be depressed below the buccal and lingual gingivæ, forming a considerable pocket between the teeth. (See Figures 189, 190 and 191.) If such cases do not receive attention, the destruction of the soft tissue will continue either as a result of the repeated impaction of food between the teeth, or from the establishment of suppuration within the inflamed tissues. Eventually, the inflammation will involve the peridental membrane at the gingival line, detaching it from the cementum, forming a pocket alongside the root. Sometimes the pressure and decomposition of food, without suppuration, will destroy the attachment of the peridental membrane. Whenever the peridental membrane is detached from the cementum by suppuration, it is the beginning of a case of chronic suppurative pericementitis.

Deposits of serumal calculus. Deposits of serumal calculus may or may not be present in these cases. Whenever there is an inflammation of the gingivæ from any cause, the quantity of serum poured out into the subgingival space is increased, and if it contains calco-globulin, a deposit may occur. As these irritations of the gingivæ are often of long duration, deposits of serumal calculus are not uncommon. As previously stated, the deposit is frequently looked upon as the exciting cause of the inflammation, while the real cause, the condition which permits the impaction of food, or which causes the irritation, is overlooked. Certainly a considerable number of cases present, in which there is no deposit.

Classification of Conditions Causing Injuries of the Gingivæ.

There are so many conditions which may cause slight inflammations of the gingivæ, that it is almost out of the question to enumerate all of them. However, it seems to be essential to a better understanding of these that an effort be made to classify them. I have, therefore, divided them into the following groups, which include the majority of causes observed:

Gingivitis due; first, to lack of contact; second, to improper contact; third, to deviations from the normal smooth contour of the teeth; fourth, to abuse of the tissues by dentists in operating; fifth, to lack of cleanliness; sixth, to misuse of tooth-picks, rubber bands, floss silk, tooth-brushes, to accidental injuries, etc. Attention has frequently been called to these injuries as a group, but it is evident that the profession does not fully appreciate the important role they play in causing peridental disease. A careful study of the following pages should serve to fix these so definitely in the mind that they will come to be looked for regularly in mouth examinations. In the plan presented for recording examinations, it will be noted that conditions falling under each of these groups may be specified. I am convinced that the dentist who will seriously undertake and follow out this plan of examination, with accurate records, will soon come to a better appreciation of conditions and will apply preventive treatment more effectively. Failures to appreciate the very severe inflammations which eventually result from lack of attention to this matter of restoring proper contacts in operative procedures, have eventually led to the loss of all of the teeth of many individuals. Certainly a very considerable percentage of the cases of diseases of the peridental membrane are due to failures to restore proper contacts. The several conditions in each group will be briefly discussed.

Gingivitis due to lack of contact of the teeth.

Separations following extractions. The teeth may be slightly separated from a number of causes. Many such cases follow the extraction of a tooth. If a first molar is extracted, for example, the second molar and second biscupid may be gradually drawn toward each other, causing slight openings of the contacts between the second and third molars, between the first and second bicuspids, and often between several others farther forward in the arch. Figure 193 shows such a case, with food impacted between the second and third molars. It is

in those cases in which this separation is slight, so that stringy foods are caught and held between the teeth, that the gingivæ are in greatest danger. In some cases the movement of the teeth continues until there is sufficient space so that food is not held between the teeth. Under such conditions the gingivæ are less liable to injury. Figure 192 shows the injury to the septal tissue resulting from an open contact between the upper lateral and cuspid.

Abnormalities of occlusion. There is a lack of contact of certain teeth in a number of cases on account of some abnormality of the position of the teeth. This may result in the impaction of food between these teeth. Such teeth usually have never been in normal contact, and, whatever space exists is not likely to change materially without operative interference.

Uneven occlusal wear. Uneven wear of cusps may cause the opening of one or more contacts which were formerly tight. A cusp of one arch will come to close with too great force between the cusps of two teeth of the opposite arch, causing them to gradually move apart a little, thus exposing the septal tissue to injury.

Weak contacts. When the pull of the trans-septal, or tooth-to-tooth fibers, is insufficient to maintain a tight contact against heavy stress of mastication, an inflammation of the septal tissue is likely to occur. This tissue may appear to be inflamed without noticeable cause. A test of the contact with the ligature will show that the teeth are in proper contact and the ligature may even pass through with a snap, yet if considerable pressure with a large instrument is made distally on one tooth, or mesially on the other, it will be observed that the contact is opened. In chewing, the stress is sufficient in such cases to force the teeth apart momentarily and crowd a few fibers past the contact against the soft tissue. This may occur several times at each meal, and even though the impacted food is removed with reasonable promptness, the inflammation and absorption of the septal tissue will usually become gradually worse. These cases are more likely to be neglected than others because of the difficulty in making out the cause of the inflammation. In mouths from which no teeth have been lost, these weak contacts are most frequently observed between second and third molars, the third molar moving distally under the stress of mastication. If any tooth in the bicuspid and molar region has been lost, the teeth next to the space are more likely

than others to move under stress, because they have lost the support of the extracted tooth.

Decays beginning on proximal surfaces. Proximal decays of bicuspids and molars, which are in normal contact, often progress unnoticed or uncared for until the lateral decay in the dentin along the dento-enamel junction has undermined the enamel of the marginal ridge of the occlusal surface. Under the stress of mastication this occlusal enamel is broken away. The enamel which formed the contact point is then lost and the opportunity is offered for food to be crowded into the cavity and wedged between the teeth. Persons who are taking reasonable care of their teeth will generally report to their dentist at once when this occurs, and the injury to the gingivæ will be temporary and of little consequence. It is in the neglected cases of this type that serious injury occurs.

Fillings or crowns which fail to make contact. In cases in which fillings or crowns fail to make contact, impactions of food and inflammation of the septal tissue results in exactly the same manner as occurs in those cases in which the teeth stand slightly apart. Figures 194 and 195 show extensive destructions of the peridental membrane, both of which apparently resulted from failures to restore proper contacts in filling operations. Sometimes it happens that, when a filling is placed which fails to make contact, the occlusion is such that the teeth move to close the contact and at the same time one or more neighboring contacts may be opened by the movement. Thus the real cause of many slightly open contacts will be found upon a careful examination of other teeth in the neighborhood, or it may even be in the opposite arch. Figures 196 and 197 are of a case in which flat proximal fillings had been placed in the distal of the first bicuspid and mesial of the second bicuspid, without restoring the contact. As a result, the second bicuspid moved forward, opening the contact between it and the first molar. There was a pocket about 4 mm. deep on the mesial side of the mesio-buccal root of the first molar. By slow separation, the second bicuspid was moved back into contact with the first molar, and properly contoured fillings were placed in the mesio-occlusal of the second and disto-occlusal of the first bicuspids, thus holding the second bicuspid in its proper position.

Gingivitis due to improper contact of the teeth.

Abnormal forms of the teeth. Contacts which are more or less broad, frequently cause a gingivitis. Between the

molar teeth, such contacts are not uncommon. Instead of presenting the normal convexity, which would give a point of contact between the teeth, the proximal surfaces may be much flattened, or the surface of one tooth may even present a slight concavity which fits more or less closely the convexity of the next tooth. As a ligature is passed through such a contact, it will drag for some distance, being held by the broad contact, instead of snapping through as it would in case of a normal contact. Shreds of stringy foods are occasionally caught between such teeth and, as time passes, this is likely to occur more frequently until the septal tissue is seriously injured. Figure 198 illustrates a case in which the distal convexity of a lower cuspid lies in a slight concavity in the mesial surface of the first bicuspid. Figure 199 shows a similar contact between the upper first and second molars. Figure 200 is of a broad, flat contact between two upper molars.

Malpositions of teeth. Contacts may be too broad on account of irregularities in positions of teeth. If a tooth is slightly rotated, or out of line, or if one may have elongated, as a result of the extraction of a tooth in the opposite arch, it may present a surface, less convex than normal, in contact with one or both of the proximal teeth, resulting in the inpaction of food.

Interproximal wear. As a result of the slight buccolingual motion of the teeth in mastication, the enamel forming the contact points gradually wears, and the trans-septal fibers of the peridental membrane draw the teeth a little closer and closer as the wear progresses, until eventually there will be a facet of considerable size on each tooth and the contact will be as large as the facet. These occur oftenest in the spaces to the mesial or distal of the first molar, in the region where the heaviest chewing is done. Food will frequently be caught between these teeth, particularly in the mouths of middle-aged and older people, although in some cases the pull of the trans-septal fibers is so strong that the teeth will be held in such tight contact that no impaction will occur. Teeth which show abrasion of their occlusal surfaces are more likely than others to also show considerable interproximal wear. Figure 201 illustrates a case in which there was extensive wear of the proximal surfaces of the molars. Figures 202 and 203 are photographic reproductions of two molars with large facets resulting from interproximal wear.

Some years ago, there came to our school clinic a gentleman

FIG. 189.

FIG. 190.

FIG. 191.

FIGS. 189, 190, 191. These sketches illustrate the injury to the septal tissue from the wedging of food between the teeth because of faulty contacts.

FIG. 189 represents the normal contour of the septum.

FIG. 190 shows the first injury resulting from slight impactions of food debris.

FIG. 191 shows the almost complete destruction of the septum.

FIGS. 192 TO 210 illustrate similar injuries from many causes. Figures 226 to 231 also illustrate injuries to the septal tissue.

FIG. 192.

FIG. 193.

FIG. 192. Photographic reproduction of a plaster model from a case in which the separation of the upper lateral incisor and cuspid had resulted in the destruction of most of the septal tissue between these teeth. This could have been prevented by the building of a proper contact.

FIG. 193. Plaster model from an impression of a case presenting with a mass of food debris impacted between two molar teeth which were slightly separated. The septal tissue was so badly injured that there was no hope that it would refill the septal space even though a good contact was made. The case should have received attention long before.

about fifty years old, a grinder of spectacle lenses. He was in some rather indefinite trouble with an upper molar tooth. The Examiner suspected a dead pulp because of the marked soreness of the tooth and redness of the tissues about it. The student to whom the case was assigned started to open the pulp chamber, but found sensitive dentin. Then I was asked to see the case. I found the tooth — an upper second molar — quite sore to the touch, and the first molar was also sore. I quickly saw that the contact points on these two teeth were worn flat so that they would readily grasp and hold food which might be pressed between them. At this time, these surfaces stood a little apart, though there seemed to be nothing between them except the inflamed septal tissue.

I asked those who were looking on to watch what I did, and taking a subgingival explorer, passed its blade along the mesial side of the mesio-buccal root of the second molar to near its apex without appearing to inflict pain. After some further demonstration of the condition, I ordered that the tooth be extracted.

The condition was the result of wedging of food between the flattened contacts. If this had received attention when it first began to catch food, the detachment of the peridental membrane could have been avoided. I found in this mouth, four other septal spaces in which the tissues were showing some shortening and other signs of injury from a similar cause. This man had no caries. I ordered cavities cut in one of the proximal surfaces in each of the four cases mentioned, and fillings made with sufficiently prominent contacts to prevent the difficulty in the future.

Improperly finished fillings and crowns. The majority of inflammations of this group are caused by improperly finished fillings or crowns. A filling or a crown with a flat contact will catch and hold food debris and cause an inflammation of the septal gingivæ in the same manner as will a flat contact of two teeth. Much too large a percentage of both fillings and crowns are imperfect in their forms of contact. We are also seeing many gold inlays, the contacts of which were probably of good form when placed, but which have quickly worn flat, because the casting was too soft to maintain its form. Thus many otherwise good dentures have been eventually lost as a result of lack of care in these operations by dentists. After one has mastered the technic of securing proper separation, there is little difficulty in making fillings which restore both proper contacts and

the full mesio-distal width of the interproximal spaces. Most dentists know how to do these things, but become careless about them, seeming not to realize the immense amount of harm that is done to the soft tissues, even though the filling or the crown may otherwise be well made. A little more time and care in these operations will serve to prevent many thousands of cases of peridental disease.

Gingivitis due to deviations from the normal smooth contour of tooth surfaces.

Sharp edges of cavities. Decays of proximal, buccal and labial surfaces, which progress until some of the enamel rods have fallen away, cause inflammation whenever the broken edge of the enamel is under the margin of the gingivæ. Decays in these positions also indirectly cause a gingivitis about several adjacent teeth, by reason of the fact that the areas will not be properly cleaned artificially by the patient, on account of the sensitiveness of the decayed areas. Such an inflammation is usually of little consequence, if of short duration, but, if neglected, suppuration may complicate the case and involve the peridental membrane.

Imperfect margins of fillings. All fillings of proximal surfaces, or gingival thirds of buccal and labial surfaces, the margins of which are not smoothly finished flush with the surface of the enamel, may cause similar inflammations. If the cavity is overfull the projecting edge of the filling will be a sufficient irritant to keep up a constant slight inflammation of the adjacent gingiva. If the cavity is not fully filled, the margin of the cavity wall will cause a similar irritation. Sufficient care has not been generally exercised in trimming to form the gingival margins of such fillings.

Crowns, bridges and partial dentures. Similar irritations are caused by crowns which do not closely fit the root end or which impinge on the attachment of the peridental membrane at the gingival line. Bandless crowns, which either project beyond the root at any point, or which fall short of being even with the root end, cause irritation in the same manner as do fillings which are too full or not full enough. Crowns with bands may cause inflammation by improper fit of the band or by any portion of the band extending so deep as to irritate or cut off the attachment of the peridental membrane at the gingival line. The tissues are thus kept in a state of irritation for years and finally a pocket is formed, or the gingivæ recede, or

both occur. It has been my observation that a large number of crown bands which may be fairly well fitted cause inflammation because the root end has not been properly prepared to receive the band. The root should be so prepared that the band, when in place, will not exceed the former size and contour of the tooth at any point.

Any portion of a bridge which is in pressure contact with the soft tissues causes inflammation, and bridges which do not actually press against the tissue, but are so constructed as to be neither self-cleansing nor easily cleaned artificially, will usually keep up an inflammation as a result of the accumulation and decomposition of food debris. Partial dentures may cause similar inflammations, either by direct pressure of clasps or of the attachments by which they are held in position.

Gingivitis due to abuse of the tissues by dentists in operating.

There has been much in our routine dental operations that has been abusive of the soft tissues, with little apparent effort to modify the injurious effects of this abuse. The tissues are bound down, lacerated and pushed out of form for varying lengths of time while making fillings. They are then released and the patient dismissed without attention, instead of washing such parts with warm water, while kneading the tissue to restore the disturbed capillary circulation.

Injuries with ligatures. One of the worst forms of this abuse is in drawing ligatures upon the crest of the arch of the attachment of the peridental membrane of the incisors on their proximal surfaces and deeply lacerating these tissues and leaving them without cleaning to inflame and to suppurate. Such treatment is unnecessarily abusive to these soft tissues, and frequently results in inflammations and suppurations of the gingivæ which subsequently involve the peridental membrane. Figures 204, 205 and 206 show the gingival lines on the proximal surfaces of two incisors and a cuspid, while Figures 207 and 208 show the proper methods of tying ligatures around an incisor tooth. Figure 287 is from a radiograph of a case in which a pus pocket on the mesial surface of an upper central incisor resulted from an injury in tying a ligature.

Some years ago, while giving a lecture on the technic of placing the rubber dam, I called for a volunteer from the class to act as the patient in the demonstrations. A young man stepped out from a front seat. He took the chair and I looked over his mouth for a moment in some amazement because of the

conditions presented. Without a word to this volunteer patient, I turned to the class and said: "This classmate of yours has suffered an injury to the peridental membranes of his incisors by the careless use of ligatures in tying the rubber dam, from which they can never recover." Then, at my request, this young man told the story of his injury to the class, and spoke particularly of the pain caused by the tying of the ligatures about his teeth. I took a subgingival explorer and demonstrated to the class the depth of the pockets which had occurred as a result of that purely mechanical injury. Several of these reached fully two-thirds the length of the roots of the teeth. Infection had occurred after the original injury and the suppuration would continue to the destruction of the membranes of the teeth, regardless of any treatment which might have been instituted at that time.

Injuries with finishing instruments and tapes. In finishing fillings, the soft tissues are often unnecessarily injured with knife trimmers and finishing files, and more especially with finishing and polishing tapes. The various polishing disks and points used in the engine also cause a share of injuries. Many similar injuries are caused by the misuse of strips and polishing devices used in the so-called oral prophylaxis treatments. Whenever the soft tissues are injured in such operations, they should be thoroughly washed and massaged to remove all particles and to restore the circulation.

Failures to remove ligatures and pieces of rubber dam. An occasional case occurs from forgetting to remove a ligature. One morning a student found a patient, for whom he had filled a cavity in a bicuspid a few days before, awaiting him with a very sore tooth. Not being able to make out the cause of the inflammation he requested me to examine the case. There was an inflammation of the gingiva which continued entirely around the tooth. Taking a small excavator I introduced it carefully into the subgingival space, and began to maneuver with its delicate point. In a short time I succeeded in catching a ligature with which the rubber dam had been tied and removed it. Rings of rubber dam are sometimes left on the teeth when the rubber is pulled away. The slender strip of rubber is often very difficult to find. A piece of rubber dam torn off and lodged in the subgingival space will often produce suppuration, and if not promptly found and removed, is liable to start the formation of a pus pocket.

Other abuses. Similar injuries are inflicted in the prepara-

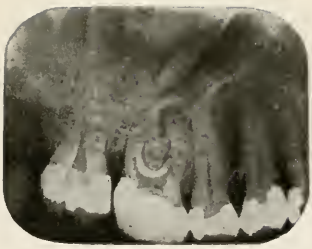

FIG. 194.

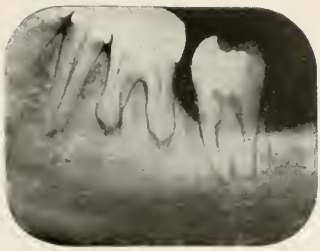

FIG. 195.

FIG. 196.

FIG. 197.

FIG. 194. Radiograph of a case in which the peridental membrane had been stripped from the distal surface of the root of an upper first molar as a result of food passing the slightly open contact. This patient had complained for weeks of the soreness of this tooth. A cavity was cut in a large mesio-disto-occlusal amalgam filling already in the tooth, and the distal contour was built out to make contact with the second molar. This stopped the injury at once, and the tooth has been comfortable since, notwithstanding the fact that the pocket remains.

FIG. 195. Radiograph of a case similar to that shown in Figure 194, but of longer standing and showing more extensive destruction of the investing tissues, so that it is doubtful if a contact could be maintained.

FIGS. 196, 197. Photographs of plaster models of a case before and after contact restoration. The patient presented with a slight pocket on the mesial surface of the root of the first molar on account of the open contact. The mesial surface of the first molar and distal of the second bicuspid were free from decay and had not been filled. The separation had occurred as a result of flat fillings in the mesial of the second bicuspid and distal of the first bicuspid. These fillings were removed, and a Perry separator was applied on several occasions to move the second bicuspid back into contact with the first molar, it being held there for a time with fillings of base-plate gutta-percha. Later, permanent fillings were made restoring normal conditions, as shown in Figure 197. It was necessary to relieve the occlusion on the distal slopes of the cusps of the second bicuspid as it was moved.

Fig. 198.

Fig. 199.

Fig. 198. Plaster model of a case in which there was an inflammation of the septal gingiva between the lower cuspid and first bicuspid on account of an abnormal form of the bicuspid, the mesial surface being slightly concave.

Fig. 199. Illustration of an abnormal contact between the upper first and second molars.

FIG. 200.

FIG. 200. Plaster model of a case with a very broad flat contact between the upper first and second molars, as a result of which the septal gingiva was injured by food impaction. Oftentimes such a contact is so tight that no food will be forced by it, and the septal tissue will not be inflamed. Treatment of such a case should depend on the condition of the septal tissue, rather than upon the fact that the contact is flat. In the case from which this cast was made a mesio-occlusal cavity was cut in the second molar, and, with a Perry separator in place, a gold filling was inserted, the proximal surface being built sufficiently convex to make a good contact. The inflammation promptly subsided.

FIG. 201.

FIG. 202.

FIG. 203.

FIG. 201. Plaster model of a case in which the contacts between the molar teeth were worn flat. As with the case shown in Figure 200, such contacts may be so tight that no food will be forced through, and the inflammation of the septal tissue should be the indication for treatment.

FIGS. 202, 203. Photographs illustrating interproximal wear. These teeth were extracted because of the destruction of the peridental membranes by the crowding of food between the teeth as a result of the flattened contact points.

tion of roots for crowns. Pressure absorption of the septal tissues, either intentionally or not, by packing gutta-percha in proximal cavities, is one of the serious common injuries. These are only types of abusive treatment of the soft tissues that do great damage. There are many others equally injurious. Every injury to the gingivæ is an opportunity for an infection which may lead to the formation of a pus pocket, and should be carefully looked after until well.

Gingivitis due to lack of cleanliness.

Lack of natural cleaning. The natural cleaning of the teeth and gingivæ by the full use of the teeth in mastication is of the utmost importance in preserving the health of the gingivæ. The scouring of the surfaces of both teeth and gingivæ by vigorous mastication prevents lodgments and accumulations, and this prevents the inflammation which would otherwise occur. Occasional cases present in which, on account of a sensitive or tender tooth, the patient has avoided for a time the use of one side of the mouth in chewing. The teeth and soft tissues of the used side will appear clean and healthy, while those of the unused side will, in marked contrast, exhibit teeth more or less coated over with lodgments of food debris along the margin of the inflamed gingivæ.

Lack of artificial cleaning. In many mouths in which the teeth are used vigorously in mastication there will be lodgments of food debris in those positions not well scoured by the excursions of food, also in any other positions, which, on account of abnormalities of position or form of the teeth, or of the gum margins, afford opportunity for lodgments. Such places require to be cleaned artificially, and whenever this cleaning is not well done, the presence and decomposition of the lodgments may cause the soft tissues to become inflamed.

Gingivitis due to errors in cleaning operations, accidents, etc.

Occasional cases are seen in which serious injury has been done to the gingivæ by the misuse of one or another of the various things used in cleaning about the teeth. The dentist should be ever on the lookout for such inflammations, and he should impress his patients with the danger of such practices and urge greater care in the future.

Misuse of toothpicks. There are two kinds of injuries caused by toothpicks: recession of the septal tissue by pressure, due to repeatedly pushing toothpicks of too large size through

the interproximal space; and inflammations caused by the rough edges and splinters of poorly made wooden toothpicks. (See Figures 209 and 210.)

I know of one case of a man of about thirty-five years, who had formed a habit of biting about an inch off the end of a wooden toothpick and pushing this piece through the various interproximal spaces with his tongue. He could start between the lower second and third molars and push the piece of wood through each interproximal space all the way around the arch, and repeat the same performance on the upper jaw, the entire manipulation being accomplished by the tongue. He had been doing this many times each day for a year or more and had caused every septal tissue to recede, exposing the proximal surfaces of all of the teeth for some distance to the gingival of the points of contact. A suppuration had occurred between a second and third molar, destroying a considerable portion of the attachment of the peridental membrane from the distal surface of the root of the second molar. In many spaces the tissue was so badly injured that a full recovery was out of the question. Many persons cause similar injuries in one or several spaces by habitually having a toothpick sticking between certain teeth. Patients should be cautioned against inflicting such injuries.

Some years ago I found so much damage being done to the septal tissues by badly made wood toothpicks that I made an outcry against their use. But since that time many of the manufacturers have so improved their methods as to remove this objection. In my wanderings, I have dropped into some of the factories where these are made, looked over the machinery and talked with those in charge, of the difficulties from both their standpoint and ours. There are still so many badly made toothpicks on the market that those buying should be careful to select only those which have been well made and the whole length carefully polished.

Misuse of rubber bands and silk floss. There is danger to the septal tissues in the use of the rubber band or silk floss in cleaning the interproximal spaces, by the snapping of the band or floss against the soft tissue, when carried past the point of contact too suddenly. A single such injury is of no consequence, but frequent repetitions will soon cause a little recession, which will gradually become worse with continued irritations.

Injuries with the tooth-brush. These are less frequent

than others, but occur often enough to require mention. The gingivæ may be injured by the use of too hard a brush, or by improper or too vigorous brushing. In both instances, the injury is most likely to be to the buccal or lingual portions of the septal tissue. In this the brush catches the margins of the gingivæ as they pass around the angles of the teeth from the buccal or lingual into the interproximal spaces, and cuts the margin away in these positions. This is done with the mesio-distal motion of the brush. I have only occasionally seen a recession of the labial gingivæ which seemed to be due to the overuse of the brush.

Accidental injuries to the gingivæ. We are liable to find stray points of inflammation, as I have sometimes called them, which can not be accounted for, except we regard them as resulting from accidental injuries. Generally such injuries last for a day or two and disappear without harm. I am convinced that some of the stray pus pockets which occur are the result of some accidental injury which has caused a suppuration that has persisted after the character of the injury has disappeared.

I have often felt that the gingivæ of the lingual surfaces of the upper incisors were very liable to be injured by accidental thrusting of foreign substances under them. Their position is more exposed to thrusts of food than any other of the gingivæ. It is certain that I have found disease beginning there which I was unable to account for under any other hypothesis than accidental injury. A few times persons have come with short length of fishbones thrust into the gingivæ, and other things of like sort. Pieces of stiff bristles from the toothbrush are also occasionally found. These things have generally been pointed out by the patient on account of some annoyance. In my observation it does not seem probable that they form any important part in the production of disease, yet occasional cases of suppuration occur.

Frequency of Different Forms of Gingivitis.

As to the frequency of the various causes of gingivitis no thoroughly conclusive statistics have been collected, although definite records have been made of a sufficient number of mouths to give at least a fair idea. Some years ago I instituted these examinations by recording the conditions found in the mouths of the senior students at Northwestern University Dental School. More recently a rather widespread examination has been made under the direction of Dr. Arthur D. Black, and

reports have been made to him of the findings of dentists in various parts of the country in the mouths of their private patients. The following quotation is from a paper read before the National Dental Association in 1913*:

"While considerably more than five hundred mouths have been examined, there will be presented at this time the summary for a tabulation covering exactly five hundred cases, selected by eliminating the cards for all persons younger than twenty or older than thirty-five years, also by eliminating cards for all persons having lesions of the peridental membrane. This summary is, then, a record of the areas of gingivitis found in the mouths of five hundred young adults, between twenty and thirty-five years of age, the average being 26.3, none of whom have disease of the peridental membrane.

"Of the five hundred mouths, twenty-five were reported as having no gingivitis, viz., just five per cent. Of these twenty-five, but seventeen had all contacts in good form. Each of the other eight had one or more open contacts, but no inflammation of the gingivæ at the time of the examination. In the mouths of the remaining 475 persons, there were reported 4265 areas of gingivitis, viz., an average of 8.53 per person for the 500 examined.

"There were 1348 areas due to deposits of salivary calculus; these were in the mouths of 198 persons, making an average of 7.8 per person. It should be stated that reports recorded deposit on more than one surface of the same tooth in 123 instances, and the very large majority of these were lower incisors marked as having exhibited deposits both lingually and labially. The percentage of the 500 patients examined who had deposits of salivary calculus is 39.6. The percentage of all of the areas of gingivitis reported as due to salivary calculus is 31.6.

"There were 563 areas reported as showing serumal deposits, in which no other cause of gingivitis was recorded. These were in the mouths of 75 persons, an average of 7.5 areas per person. The percentage of the 500 examined who had deposits of serumal calculus is 15. The percentage of all of the areas of gingivitis reported as having serumal deposits is 13.1.

"There were 33 mouths in which deposits of both salivary and serumal calculus were recorded, making a total of 140 per-

* Something of the Etiology and Early Pathology of Diseases of the Peridental Membrane, With Suggestions as to Treatment. Arthur D. Black, Dental Cosmos, Vol. LX, 1913, p. 1219.

FIG. 204.

FIG. 205.

FIG. 206.

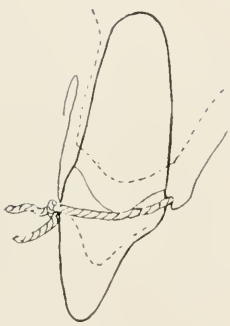

FIG. 207.

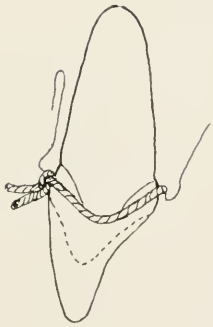

FIG. 208.

FIGS. 204, 205, 206. Upper central incisor, upper cuspid, and lower incisor, showing curve of gingival line on proximal surfaces.

FIG. 207. Diagram showing the relation of the ligature to the attachment of the soft tissue at the summit of the arch as it passes labio-lingually.

FIG. 208. Illustration of the position of the ligature in its relation to the interproximal attachment of the soft tissues to the tooth. If this is firmly drawn and pushed to the gingival as far as shown, the cutting of the attachment of the soft tissues to the neck of the tooth is inevitable.

FIG. 209.

FIG. 210.

FIG. 209. Plaster model of case in which the septal tissue between the second bicuspid and first molar had been seriously injured by the use of a wooden toothpick. The toothpick had been repeatedly pushed entirely through the interproximal space from buccal to lingual, and had forced the soft tissue farther and farther away from the contact. This patient was directed to use a rubber bulb syringe after each meal, to cleanse this and other spaces.

FIG. 210. Plaster model of lower front teeth. The primary recession on both the lingual and labial was due to deposits of salivary calculus. Later the patient formed the habit of pushing a wooden toothpick through the spaces between the teeth and caused the septal gingivæ to recede.

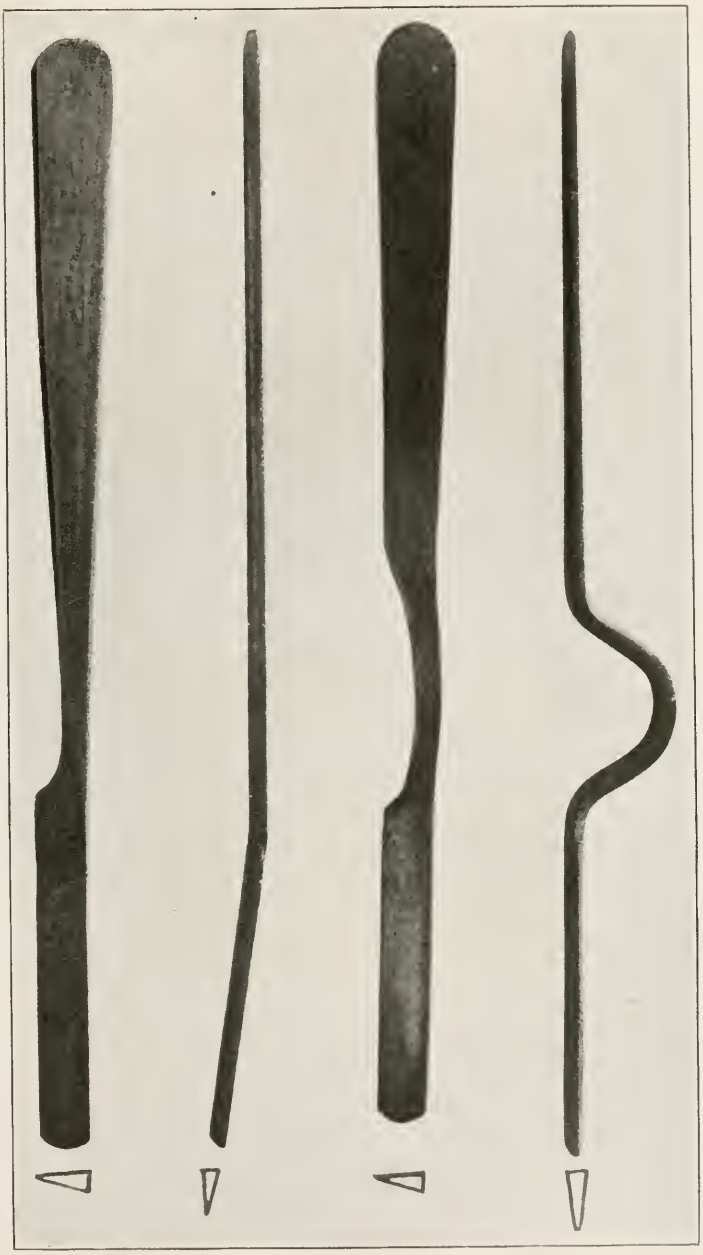

FIG. 211.

FIG. 211. Four files such as were formerly used for separating teeth and finishing proximal gold fillings. The cross-section diagram below each file gives an idea of the space which was left between the teeth when such files were used.

Fig. 212.

Fig. 213.

Fig. 214.

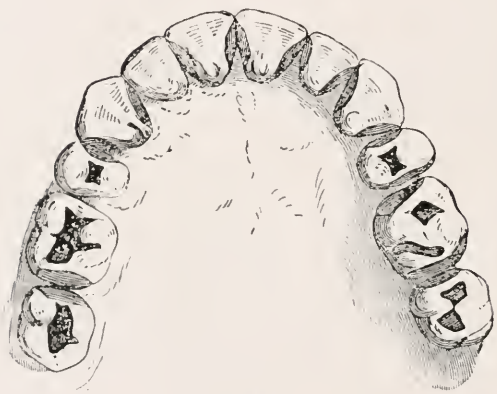

Fig. 215.

Figs. 212, 213, 214. Diagrams to show results of the use of the old finishing files illustrated in Figure 211.

Fig. 212 shows about the normal contour of the teeth from a buccal view.

Fig. 213 shows the spaces left after the use of the files.

Fig. 214 shows the spaces after the teeth had moved forward until they were in contact near their gingival lines.

Fig. 215. Reproduction of an illustration in Dr. Robert Arthur's book, "Treatment and Prevention of Decay of the Teeth," published in 1871. The dark lines on the proximal surfaces of the teeth indicate the portions which, it was advised, should be cut away, fillings being placed from the direction of the lingual embrasure. This method was suggested as a substitute for the former plan of cutting from the entire proximal surface.

sons having either kind or both. This leaves 360 of those examined for whom no deposits were reported.

"There were 2354 areas of gingivitis due to other causes than deposits, subdivided as follows: 783 were due to bad margins of fillings or crowns, 496 to lack of contact of proximal fillings or crowns, 305 to improper contact of proximal fillings or crowns, 263 to malpositions or atypical forms of proximal surfaces, 255 to lack of contact of teeth having no caries of proximal surfaces, 233 to caries of proximal surfaces, and 19 to worn contacts. If the areas due to malpositions, etc., to lack of contact of undecayed teeth, to caries, and to worn contacts, are not counted, there remain 1584 areas, the large majority of which are due to imperfect dental operations. These are more than 37 per cent of all areas of gingivitis reported. The percentage of all areas of gingivitis reported as due to trauma is 55.1. For all persons included in the tabulations there is an average of 4.7 areas of gingivitis per person due to trauma."

It will be observed that 95 per cent of all persons included in the tabulation had one or more areas of gingivitis and that the average number of areas per mouth was 8.53. This statement alone should impress the necessity for greater care in the examination of cases and the institution of procedures for the prevention or cure of such inflammations. It will also be noticed that less than one-third of the areas of gingivitis were caused by deposits of salivary calculus, and less than one-sixth by deposits of serumal calculus, more than half of all areas being caused by injuries and irritations. Attention is especially called to the fact that 1584 areas out of the total of 4265, or about 37 per cent, were reported as due to imperfect dental operations.

History of the Attitude of the Profession Toward Injuries of the Gingivæ.

The changes in the attitude of dentists toward the preservation of the gingivæ form an important element in the development of dentistry. From a subject in which no interest was manifested, it has become one of almost vital interest to all the better class of men of the profession.

For many years after I entered practice, almost no attention was given to this subject by dentists within my acquaintance. This was not very wide, however, at that time. But as I now look back over that period, I may say that my acquain-

tance with dentists, and with what they were doing and striving for, developed with fair rapidity.

The use of noncohesive gold and the filing of V-shaped spaces. The profession was then using noncohesive gold, with which it was practically impossible to restore the form of a proximal surface of a tooth, the occlusal surface of which had broken down at the position of the marginal ridge. These fillings required a different standard of cavity preparation from that now in use. For instance, if a proximal cavity in a molar tooth was so large that a part of the occlusal enamel was broken in, forming what we now know as a mesio- or a disto-occlusal cavity, a file was first used with which a V-shaped opening was cut between the two teeth of such width that the break in the occlusal surface was cut out. In this case, if the tooth which had been in contact with the decayed one was not decayed, the cutting was done with a safe-sided file so that the surface of the sound tooth would not be cut. If it had also a considerable cavity, both teeth were cut in a form which produced a large V-shaped space between them. Four of these files are shown in Figure 211, and an idea of the spaces cut by them may be had from the diagrams, Figures 212, 213 and 214.

The cavities were formed in the flat surfaces produced by this process of filing. They were filled and then finished with the same file with which the cutting of the teeth had been done before forming the cavity. The filling was finished flat and level with the cut surface of the tooth. This left a broad V-shaped space between the teeth with the acute angle of the V toward the gingiva. In this space food would wedge in chewing, but the space so formed was so broad and opened so widely toward the occlusal that food generally did not remain impacted.

In chewing any such foods as meats, especially beefsteak, pressure came directly on the gum septum, if there was any left after the operation, and produced a great deal of pain. This usually continued until the gum septum was destroyed. The teeth often dropped together, coming into contact near the gingival line. Then food would occasionally be forced past this contact and lodge. For this there seemed to be no remedy in sight, even when the difficulty was correctly diagnosed, which was done by but few men. Such cases usually brought on suppuration. Then inflammation spread, other contacts loosened, food lodged in them also, and the occlusion went astray, so that the soft tissues became generally involved. Many of the cases

ended in the extraction of the teeth and the substitution of artificial dentures.

In filling teeth not so much decayed, the habit of the time was to cut with a flat separating file to secure sufficient space to make the filling, and then to finish the fillings flat with the same file with which the teeth had been separated. This left a narrow space with flat sides which caught food and held it so that it was forced upon the gum septum, and in many cases destroyed this tissue. This was the worst form of cutting for cavity preparation ever practiced; although it did not apparently mutilate the teeth so much as the broad V-shaped cuts, it brought about greater destruction of the peridental membrane.

In spite of these conditions, some patients seemed to so manage, by certain cleaning processes which each would devise for himself, to keep the investing tissues in fairly good health and retained their teeth for many years. Also, there were some dentists, wiser or more skilful than the rest, who so performed their operations that these injuries were obviated in greater or less degree. The means which they employed have long ago become inapplicable, because they are supplanted by better methods.

Discovery of the cohesive property of gold foil. The cohesive property of gold foil was discovered and reported* in 1855 by Dr. Robert Arthur, who was then teaching Operative Dentistry in the Philadelphia College of Dental Surgery. This discovery was destined to place dentists in position to do away with the cutting of the V-shaped spaces between teeth, and to build contacts which would restore the teeth to normal form, so that they would protect the septal gingivæ. But the use of cohesive gold to the best advantage for this purpose was very slow in its development.

It required much time for men to learn to use the welding property of gold successfully. For this property was menaced and destroyed, often permanently, by substances and forces not visible to the eye, or that could be told by any other test than the trial of the gold. Some years later I made a careful study of the development and loss of the welding property of gold. In fact, I read my first paper† before a dental society on this subject.

* Dental News Letter, Vol. 8, p. 131.

† Gold Foil. Paper read before Illinois State Dental Society, published in Missouri Dental Journal, 1869, p. 283. This paper was subsequently revised and read before the New York Odontological Society in 1874, and was published in the Dental Cosmos, Vol. 17, 1875, p. 138.

The tendency after the discovery of the cohesive property of gold was to continue the old forms of cutting the teeth for the preparation of cavities. Dr. Arthur published a small volume, "A Treatise on the Use of Adhesive Gold Foil," in 1857. In this volume there is no hint of restoring proximal surfaces to the normal form. In this condition of the mental conceptions of dentists, there could be no improvement in the protection of the septal tissues. This was not altogether because of failures to recognize these conditions, as will be seen by the record of the struggles of many men to remedy this evil. Dr. Robert Arthur himself later wrote a book* in which he introduced a different form of cutting proximal surfaces in the effort to prevent these injuries. An illustration in this book is reproduced by Figure 215. The plan was to cut a V-shaped opening looking to the lingual, instead of the occlusal, leaving an angle of the enamel on the buccal or labial side to make contact, and in this way protect the septal tissues. This form increased the difficulties of cutting the spaces and the insertion of the fillings was not so convenient. Under these conditions very little use was made of the new forms proposed.

The wooden wedge. Then there came a period of the wildest indifference to the septal tissue. This was the era of wedging teeth apart by driving wooden wedges between them. Most men were learning to manipulate cohesive gold before the rubber dam had come into use. The wooden wedge was used primarily to obtain some separation of the teeth in order that the cavity could be prepared and filled with less mutilation of the tooth with the file. The wedge was made to serve another purpose also, which was really the more attractive feature. The soft tissues were carried away from the cavity margins by the wedge, making it much easier to keep it dry while packing the gold. I have often seen these wedges sticking out to both buccal and lingual, with small cotton rolls lodged against them, and these backed up by larger rolls of cotton, in order to keep the cavity dry. When the filling was finished there was little or no septal tissue left between the teeth. In many of the cases, every bit of this tissue was either crushed out or so mutilated that it sloughed away. In those days, I frequently used this plan myself and carefully observed the results in the extended suppurations of the peridental membranes. I also saw an abun-

* "Treatment and Prevention of Decay of the Teeth." 1871.

dance of this among the patients of other dentists. Really, this was creating havoc of the soft tissues.

Discovery of the rubber dam. The rubber dam was discovered and made available for protecting cavities from moisture in 1864. It was the suggestion of Dr. S. P. Barnum, who spent very much time in his own office in showing dentists who visited him how to apply it. The difficulties men have experienced in learning to use the rubber dam with ease and facility would be a very amusing feature to me if it were not really so serious. So many men in practice and so many students in our schools seem to have great difficulty in mastering the technic of the application of the rubber dam, although it is generally very easily and quickly done by those who once get the knack of it.

These difficulties held back for many years the general use of this best of all means of keeping cavities dry. Notwithstanding the development of the many helps we now have in its use, many men in practice do not really handle it well. For this reason I endeavored in my work on Operative Dentistry to so standardize the necessary movements as to make the matter very simple.*

Physiological importance of tooth forms. During all of this time, and on up to recent years, there was little appreciation among dentists of the physiological importance of tooth forms or of the relation of these forms to diseases of the gingivæ. There was no book on dental anatomy from which any accurate information could be gained regarding the forms of the teeth, so that the pathological results which might arise from faulty forms could be recognized. For a year or two before I wrote my book on Dental Anatomy, in 1891, I was in the habit of asking prominent dentists questions relating to the anatomy of the teeth. One question frequently asked was: "What is the difference between the cusps of the upper first molar and lower first molar?" I found almost no one who was able to answer this question, or even to give the number of cusps on each of the teeth. The common answer was: "Show me the teeth and I can tell the one from the other." They could distinguish an upper molar from a lower molar in extracted teeth, even when the roots were hidden from them, with unerring accuracy, but to tell what the elements of difference were, was another question entirely. Ignorance of the special forms of

* Black's Operative Dentistry, Vol. 2, p. 69.

the contact points for the prevention of lodgments was profound. In most of the dental schools this subject is now taught, and the general knowledge of the dentist has been wonderfully increased within the last twenty years.

In this mental attitude toward tooth forms men began the effort to restore the lost forms of teeth with cohesive gold. The result was a most miserable failure at first, and seemed to gain in accuracy very slowly. The facts became clear enough as I followed and studied the results of filling operations by McKellops, Webb and others of the best men of the past, in the manipulative sense, whom the world has known. I studied their reproductions of form and the results of these betterments in the protection, or failure of protection, of the soft tissues investing the teeth. The manipulation was fine. These men, and many others who approached their manipulative skill most closely, could produce any form they desired. The difficulty was not in their power of manipulation, but was because of failure to gain a correct conception of the form required. As our schools have advanced in teaching the anatomical forms of the teeth, so has the profession gradually advanced in its conception of the forms which are necessary to give the investing tissues the best protection.

TREATMENT OF GINGIVITIS DUE TO INJURIES.

ILLUSTRATIONS: FIGURES 216-225.

Most cases of gingivitis due to injury may be cured by very simple means. There is little in the treatment of any of these conditions which may not be accomplished in a comparatively short time. The difficulty is not so much in the technic to be employed, as in the fact that dentists generally have not come to recognize these inflammations as forerunners of chronic suppurative pericementitis. Many men seemingly are so blind to this relationship that no consideration whatever is given to inflammations of the gingivæ.

The gingivitis and pericementitis should be clearly recognized as different stages of the same disease. As previously mentioned, the connection between the two has not been appreciated, by reason of the fact that the progress is so slow and the elapsed time between the beginning gingivitis and the establishment of an easily recognizable pocket is so great that the two are not associated.

The mouth examinations which have been tabulated reveal the somewhat startling fact that there are areas of gingivitis in the mouths of about ninety-five per cent of all adults and that these areas average more than eight to each mouth. Can the dental profession go calmly on with its daily routine as heretofore, paying little attention to these inflammations, while it is at the same time actually producing a very fair percentage of the whole number by lack of consideration for the gingivæ in operations performed? The pathology of these lesions is comparatively simple; it should be fully understood by every dentist. It must become part of the routine examination of the mouth to record the inflammations of the soft tissues and to give them the most careful study and treatment in order that the health of the mouth and of the individual may be conserved.

Really, the thought which should be constantly uppermost in the mind of every dentist in the consideration of the treatment of these conditions is that these areas of gingivitis may in most cases *be prevented* by the same simple treatment which is employed to cure them when they have occurred. The dentist should, therefore, come to recognize clearly those conditions

which cause these inflammations and do whatever may be necessary to prevent them. In the entire field of dentistry no service is of greater value. If each dentist will bring himself to realize that he may be preventing the loss of the entire denture every time he prevents or cures a slight gingivitis, he will come to really appreciate the value of this service.

The technic to be employed in the individual case is usually determined when a proper diagnosis is made. If the inflammation is due to an open contact, it is obvious that an operation should be performed which will restore such a contact to normal. If a contact is not in proper form, the malform should be corrected, whether it be of the tooth or a filling. If the patient is, by neglect of proper care, permitting an inflammation, he should, if possible, be made to take an interest in his own welfare to the extent that the difficulty will be remedied. If he is causing inflammation by improper use of toothpicks, rubber bands, etc., his attention should be called to such errors. Each case is likely to present, on careful study, a little problem in itself. The condition of the entire mouth must often be taken into consideration. Oftentimes the cause of an inflammation in one jaw will be found in the opposite jaw. Some suggestions as to the various forms of treatment may prove of interest.

In cases of lack of contact. If two teeth are slightly separated on account of the extraction of a neighboring tooth, a careful examination of the occlusion should be made to determine whether or not the movement is apt to continue. If so, then it may be best to make no effort to restore the contacts. If, on the other hand, certain contacts are open just enough to catch stringy food and thus keep up a gingivitis, and there is not the probability that the separation will continue to the point where food would not be held between the teeth, the contact should usually be restored by a filling operation. If the open contact has been caused by some abnormality of occlusion, the abnormality should, if possible, be corrected. In some cases, and particularly in older people, it will be best to restore the contact in some other way, either by grinding a little off those cusps which are holding the teeth apart, or by placing a filling. If the occlusal wear of the teeth has caused the opening of a contact, a little grinding of the cusp in the opposite arch will usually be sufficient, as this will permit the teeth to be moved back into contact by the pull of the trans-septal or tooth-to-tooth fibers.

In restoring contacts by filling operations, a careful study of the widths of the various interproximal spaces should be

made. Sometimes it will be best to cut a cavity in an undecayed tooth, and place a filling which will be built out to restore a tight contact in proper form. Generally, however, some other plan may be followed. If there is a proximal filling in a neighboring tooth, it may be replaced or built out and the contact made sufficiently prominent to close the open contact. For example, if the septal tissue between the first and second bicuspids is inflamed on account of an open contact, and there are no decays and no fillings in either tooth, but there is a mesio-occlusal filling in the first molar, this filling may be replaced with one having a more prominent contact, a separator being used to force the second bicuspid close against the first bicuspid. Or, if the previously placed filling in the molar is of considerable size, a small cavity might be cut in this filling without removing it, and a filling placed, accomplishing the same result.

Weak contacts may be treated in the same way, either by making the contour of the proximal surface of one of the teeth more prominent, or by building out a filling in a neighboring tooth.

If there is only a gingivitis, these cases usually do well. If, however, the case has progressed until the peridental membrane has been detached, the difficulty of curing the case by building out a contact is greatly increased, as will be mentioned in detail later. Very often the weak contacts occur secondarily to some trouble with a next neighboring contact, and both may be made right by the operation required for the one. The loss of the mesial surface of a second molar by caries may result in a mesial movement of the tooth, thus permitting food to leak through the contact between the distal of this tooth and the third molar. The restoration of the mesial surface should be so made as to restore both contacts.

If a previously placed filling has failed to restore the contact, it should be either replaced or built to form by cutting a small cavity in it and filling this. A gold crown may sometimes be built out in the same way, by cutting a cavity and placing a filling in it, although it will usually be necessary to replace such a crown. Sometimes the near-by teeth may be moved to make contact with a crown, by fillings which may be required in them.

Danger of disturbing the occlusion. In all of the above, the occlusion must be carefully watched. After each operation, the patient should be requested to close the teeth a number of times, while a careful watch is made for movements of the teeth when the occlusion is pressed hard, also when it is relieved, to

see if any contacts are opened and closed in this way. The movement of the teeth necessary to restore proper contacts in one arch, may lead to a disturbance in the opposite arch, if care is not exercised. If any cusp strikes more noticeably than the others, enough should be ground off to relieve it. Otherwise the readjustment of the teeth which would occur within the next few days might result in the opening of some contact. It will usually be necessary to grind a little from some particular portion of the slope of the cusp, and the occlusion should then be examined again and again to see that this is properly relieved. This grinding should not disturb the cusp as a whole. It should never be necessary to grind down an entire cusp.

For example, if the lower bicuspids were slightly separated on account of a flat mesio-occlusal filling in the first molar, and a separator should be placed between the second bicuspid and first molar, for the purpose of moving the second bicuspid hard against the first bicuspid and holding it there by building out a sufficiently prominent contact on the first molar, it might be that the mesial movement of the second bicuspid would cause the mesial slopes of its cusps to strike too hard against the distal slopes of the cusps of the upper first bicuspid. If this were not corrected at once, the tendency would be for the upper first bicuspid to be moved mesially, possibly enough to weaken the contact between it and the upper second bicuspid. Or, the lower second bicuspid might be moved distally enough to reopen the contact between it and the lower first bicuspid. The least bit of grinding, which may usually be done with a disk, from the mesial slopes of the cusps of lower second bicuspid or the distal slopes of the cusps of the upper first bicuspid would prevent such a disturbance of the occlusion.

In cases of improper contact. If there is an inflammation of the septal gingivæ due to too broad a contact of two teeth, there is usually but one plan of treatment which will be effective; a cavity must be cut in one of the teeth and a filling placed which will be more convex than was the surface of the tooth, thus making a rounded *contact point,* which broadens the tooth mesio-distally and will so touch the contact of the approximating tooth that it will not catch and hold food debris.

Malposed teeth which form improper contacts can in some cases be moved so that proper contacts will be restored. Sometimes it will be best to cut cavities and make fillings of such form as to remedy the condition.

Contacts which have become flattened by interproximal

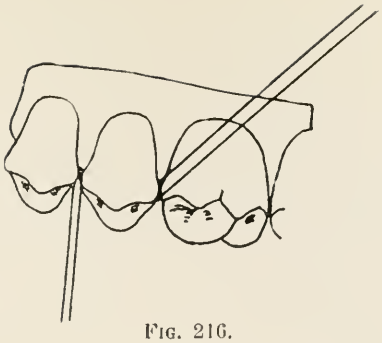

Fig. 216.

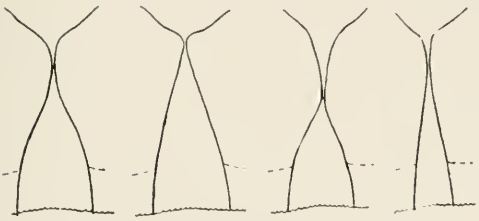

Fig. 217.

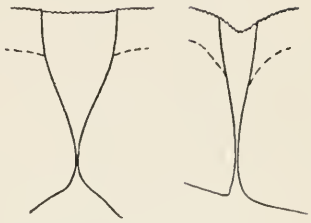

Fig. 218.

Fig. 216. Method of testing contacts. In testing a contact the ligature should be first carried through from occlusal to gingival, then the two ends of the ligature should be held parallel in the occlusal direction, as shown between the two bicuspids; the distance between the strands will indicate the bucco-lingual width of the contact. The two ends should then be held parallel in the bucal direction, as shown between the second bicuspid and first molar; the distance between the strands will indicate the occluso-gingival width of the contact. In either position, if the parallel strands are more than from 1½ to 2 mm. apart, the contact is too broad.

Fig. 217. Good and bad forms of proximal contacts, buccal view. The proper position of the contact point for bicuspid and molar teeth is about as shown in A, just a little to the gingival of the marginal ridges. The convexity of both surfaces at this point should generally change to a slight concavity in the gingival direction.

The position of the contact in B is too far occlusally. With such a form of contact, less than the normal portion of food will pass through the embrasures, and the exposed portions of the proximal surfaces of the teeth will not be normally cleansed in mastication.

The position of the contact in C is too far gingivally. The long approaches from the occlusal invite the wedging of food between the teeth, with the danger of forcing the contact open.

The position of the contact in D is about right, but the teeth have not been separated to restore the normal mesio-distal width of the interproximal space.

Fig. 218. Comparison of anterior and posterior interproximal spaces, labial and buccal view. It will be noticed that the base is much wider mesio-distally between the bicuspids and molars than between the incisors. This would seem to give the more support to the septal tissue between the back teeth. It should be remembered, however, that the line of attachment of the gingivæ to the incisors is very convex, extending far toward the incisal on the proximal surfaces, thus compensating for the narrower alveolar base.

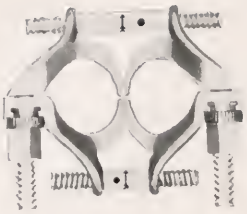

Fig. 219.

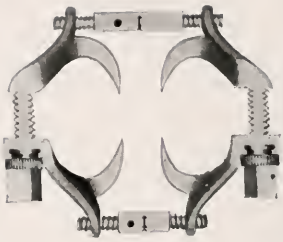

Fig. 220.

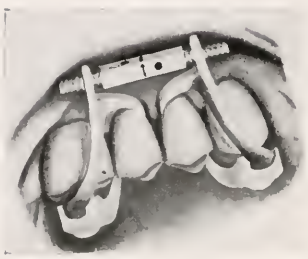

Fig. 221.

Fig. 222.

Fig. 219. The Universal separator as arranged for the smallest teeth, or with the points closed together in both directions.

Fig. 220. The separator opened full width bucco-lingually by turning the adjustment nuts. By means of these nuts the adjustment for the fitting of any size of tooth is made before the separator is applied. It is also opened a little more than half its full width mesio-distally by turning the separating bars. After the separator has been adjusted to the teeth, these separating bars are turned until sufficient separation is made.

Fig. 221. The separator applied to the bicuspids. In this case the points or claws impinge on the soft tissues at the necks of the teeth; it must be loosened at once and the separator propped up, as shown in Figure 222.

Fig. 222. The separator is propped up with gutta-percha, modeling compound or other suitable substance placed on the occlusal surfaces of the teeth under the adjustment bars, preventing the separator from slipping gingivally and injuring the gums. The teeth are shown separated.

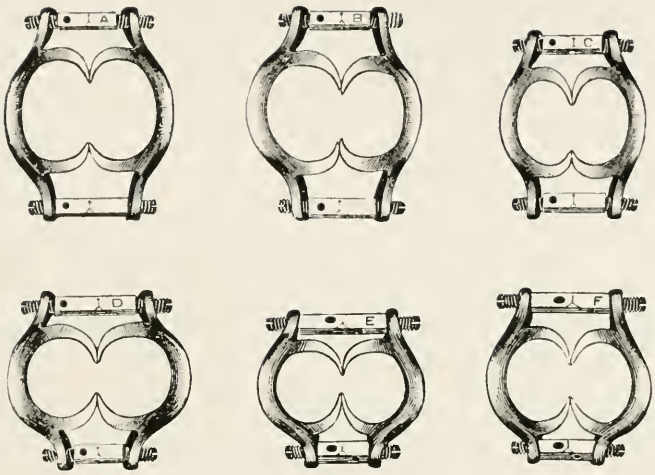

FIG. 223.

FIG. 223. The regular set of six Perry separators. It will be observed that these do not have the screws for bucco-lingual adjustment. It is therefore necessary to have several instruments to fit the various teeth. The instrument marked A on the separating bar will generally fit between any two molar teeth; the B between the second bicuspid and first molar; the C between the bicuspids; the D and E between the incisors, the D being for longer teeth than the E; the F between small bicuspids.

These instruments are to be preferred to the "Universal" shown in Figures 219 and 220, as they are more easily placed, are steadier and much less in the way.

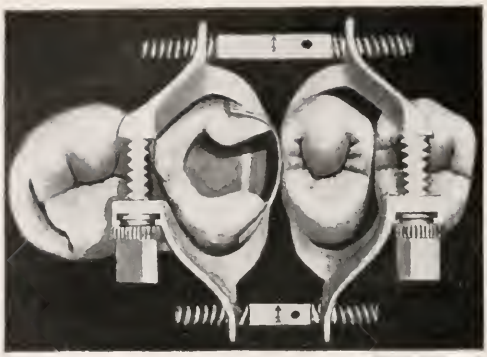

FIG. 224.

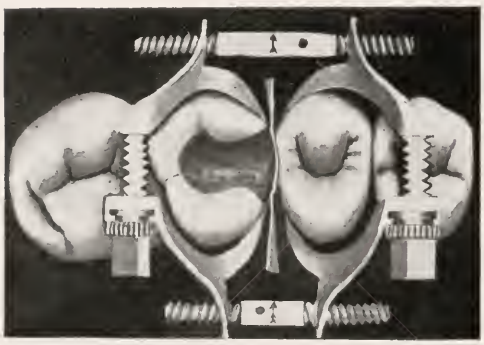

FIG. 225.

FIG. 224 shows the matrix for an amalgam filling held in position by the separator. This form, which is quite fully explained by the illustration, is a good and efficient method of using the combination of the two instruments. When the separator has been placed on the teeth and tightened just enough to stay well in place, the matrix is easily forced between the tooth and the claws of the separator and is then firmly held and tightened against the tooth. Or the matrix may be tied in place and the separator applied later.

FIG. 225. When the filling has been placed and the occlusal surface trimmed to form, and some time has been given for the amalgam to become hard, the separator is loosened and removed; then the ends of the matrix are straightened out to the buccal and lingual, as shown. The separator is then replaced and tightened until the grasp of the two teeth on the matrix is loosened. The matrix is then removed and the proximal portion of the filling trimmed to form. Finally the separator is removed.

wear must be treated the same as teeth having abnormally flat contacts; the proximal surfaces must be made sufficiently convex by filling operations. Oftentimes the placing of one filling with a prominent contact in the bicuspid and molar region will be sufficient to tighten all of the contacts of the side so that impactions will be prevented. In keying up the arch, the fact must be kept in mind that the interproximal wear of the teeth will shorten the distance around the arch from one third molar to the other third molar about one-third of an inch by the time a person is forty years of age. Of this wear, much the greater part occurs in the bicuspid and molar region, where the heaviest mastication is done.

The most frequent cases of flat contacts are fillings and crowns. Most such fillings and porcelain crowns can be trimmed to proper form by placing a separator for a moment to get room for the finishing. This is, of course, what should have been done when the operations were made originally.

Other conditions. Little need be said regarding the treatment indicated in those cases in which a cavity edge or a filling margin is causing an irritation of the overlying tissue. If sharp edges of cavities are causing inflammation, the placing of proper fillings will remedy the trouble. If filling margins are not smooth, they should be made so by trimming if overfull; by adding to or replacing fillings which are not full enough. Crowns which do not properly fit the root end, or which for any other reason cause inflammation, should often be removed and new crowns made. Any procedure should be undertaken which promises to permit the gingivæ to return to a healthy condition.

More careful study of cases necessary. The successful treatment for the prevention or cure of the cases of gingivitis which are constantly presenting, involves the more careful study by the profession of all of these slight inflammations. The discovery of each area of inflammation should lead to the immediate investigation of the cause and to the operation necessary for its correction. By following this plan, each operator will soon come to have a better understanding of the results of his errors in technic, of his neglect of some little thing, of his abuse of the tissue in the application of the rubber dam, in finishing and polishing operations, etc., so that he may avoid these in the future.

This abuse of the gingivæ during operations in the management of dental caries has been common ever since I have been in practice. It has become so fixed in the minds of dentists that

it will require the combined and long-continued efforts of all of the best men in practice to eradicate it. Many years of effort have been expended to bring the filling of cavities of decay to the present standard of efficiency, and many more will be required to bring a majority of dentists up to the efficiency of the more skilful men of the present time. The correction of the abuses of the gingivæ will necessarily follow a similar slow development. This should not be regarded as a discouragement. The accomplishment will come in gradual improvements, and will come with time.

In this the most rigid rules of looking after what is now regarded as the minor details of practice will be necessary. Every injury to the gingivæ should have a place in the record of examinations. The treatment of these and the outcome of each case, as determined by later examinations, should be recorded. In this way men may soon have records of these matters which will be of great practical value and many serious cases of disease will be avoided.

In a similar way dentists must study those cases in which patients are permitting or causing inflammations, either by lack of care in cleaning operations or by errors in their efforts. These will be considered in detail under the discussion of Mouth Hygiene. It need only be added here that it should be a part of the dentist's duty to carefully scrutinize the mouth of each patient at regular intervals for the particular purpose of noting and pointing out areas of inflammation which may be eliminated by better care by the patient. The dentist must put himself into this work with an earnestness which will command the interest and coöperation of the patient.

An exact method of separation essential to success in building proper contacts.

As early as 1846,* possibly much earlier, rubber wedges were used to separate teeth to gain space for the placing of proximal fillings in the front teeth. As has been mentioned, it was the common practice to drive wooden wedges between the back teeth for the same purpose fifty years ago. Both methods were used for many years, and the rubber wedge is used to a considerable extent yet. The wooden wedge was both very painful and terribly destructive to the septal tissues. The rubber wedge often caused the teeth to become painfully sore as the

* The Surgical, Mechanical and Medical Treatment of the Teeth, by James Robinson, second edition, 1846, p. 107.

result of the continuous pressure, and the soft tissues were frequently injured. The soreness of the teeth occasionally made it necessary to postpone filling operations, or if they were undertaken while the teeth were sore, much pain was inflicted and oftentimes the operations could not be properly performed. Other methods were unsatisfactory for one reason or another. People came very justly to have a horror of the separation of the teeth.

To-day we separate teeth principally for the purpose of restoring proper contacts. With our present methods of cavity preparation, separation for convenience of access is generally unnecessary for fillings in the back teeth, and only occasionally in the front teeth. Therefore, only a little movement of the teeth is necessary — just enough to permit of trimming and polishing and still leave a proper contact. It is understood that if the teeth have moved together more closely than they should normally be, additional separation is necessary. Several forms of contacts and a method of testing the widths of contacts are shown in Figures 216, 217 and 218.

The Perry separator. In 1879 Dr. Stafford G. Perry* invented a separator which is so designed that the teeth may be lifted apart without the slightest injury to the soft tissues and with little or no discomfort to the patient. Separation may be gained at the same sitting at which the filling is placed, and in the placing of gold fillings, the appliance really makes the operation easier for the patient by holding the tooth being filled tightly against the next tooth in the arch, thus gaining its support in resisting the force of the mallet. In placing amalgam fillings, the separation is gained and the matrix held tightly at the gingival by this appliance. For the setting of inlays, the separator is not always necessary, but there is often a decided advantage in lifting the teeth apart just a little. The Perry separator and its application are illustrated in Figures 219 to 225.

It is not within the scope of this book to do more than emphasize the imperative need of serious study of the best methods necessary to proper contact restoration. The Perry separator is the best instrument that I know of for this purpose to-day. This, or some similar appliance having four jaws which may be set against the teeth without impinging on the soft tissues, should be in the equipment of every dentist and should be used for practically every proximal filling operation. In con-

* Dental Cosmos, Vol. 21, 1879, p. 253.

nection with the placing of amalgam fillings, it should be applied when the filling is placed and again when it is polished. Satisfactory contact restorations can not be made otherwise.

The study of contact forms of both teeth and fillings should be a constant one by every dentist. In the examination of each mouth every contact should be tested and a record made of those which are not normal. If the contact is not right, there will usually be an inflammation of the septal tissue which can not recover until the faulty contact is corrected. In this constant care of contacts lies, in considerable measure, the secret of the prevention of peridental disease.

Dentists who have not mastered the technic of the use of the separators of the Perry type should begin at once the effort to do so and continue until they can easily place these separators in practically any position in the mouth, without serious discomfort to the patient. The technic of application is given in the descriptions of the illustrations, Figures 219 to 225.

This separator catches the teeth near the gingivæ with its jaws, and the teeth can be separated to any extent that may be desired, by the slow motion of the screws on the sides. Teeth which are firmly set, as the molars, will often require that considerable force be applied in turning the screws. Attention is called to the fact that the spring in the jaws of the separator will exert a constant pull on the teeth and may cause some additional movement within a few minutes. If the separator is applied during the latter part of the cavity preparation, and, if the spring of the separator during the placing of a filling has not carried the teeth far enough apart, a little further tightening of the screws will give space for the final finishing of a gold filling. Single-rooted teeth move more easily than the molars and care must be taken in tightening the screws upon these, for they may be carried much farther apart than is desirable. One should acquire a very accurate conception of the force required to move the teeth with such a separator and learn to use only the amount of force necessary in the individual case. For the patient, this mode of separation is the easiest that has ever been devised. The separation is made and the operation completed at one sitting. There is no injury to the membranes about the teeth and the teeth are not sore, as they often are with other methods.

After the teeth are allowed to drop on their new contacts, and the occlusion is carefully examined and corrected, if need be, as has been directed, no discomfort of consequence should

be felt and mastication may go on with these teeth immediately afterward.

To accomplish all of this, however, requires the development of skill in the application of the separators, the prevention of injury to the gingivæ by them and the use of the proper force in the tightening of the screws in each case.

It will be of interest to refer to one case in which it was necessary to secure considerable movement of the teeth in order to restore a proper contact and the full mesio-distal width of the interproximal space. A young lady who had been a patient of mine since childhood, left me and went to her husband's dentist after her marriage. Several years later she returned complaining of soreness of the tissue between a second bicuspid and first molar. There was a mesio-occlusal filling in the molar which was flat on the mesial surface and did not quite make contact with the bicuspid, and the space was packed with food at each meal. The molar had moved mesially so that it was necessary to gain considerable space to restore the normal contour of the tooth.

The filling was removed and a Perry separator was applied. Pieces of gutta-percha were warmed and placed between the occlusal surfaces of the first bicuspid and second molar and the bows of the appliance where these crossed over these surfaces from buccal to lingual. (See Figure 222.) The gutta-percha was allowed to harden before the separating screws were tightened. This use of gutta-percha is particularly important, as the jaws would otherwise be inclined to slip in the gingival direction with the increase of the pressure, and thus not only injure the gingivæ, but cause unnecessary pain. Then the screws were tightened slowly until it was thought that as much pressure was put on as was prudent to use. The cavity was then filled with base plate gutta-percha, which was packed solidly against the distal surface of the second bicuspid. In doing this an instrument was placed between the teeth to the gingival of the gingival margin of the cavity and was held firmly in place while the gutta-percha was built over it; the instrument was then removed. By this means the crowding of the gutta-percha upon the septal tissue was avoided.

After the gutta-percha had hardened, the separator was removed. Thus the gutta-percha held the space which had been gained by the separator. The patient was directed to use this tooth as much as possible in mastication and to return four days later, at which time the same separator was placed, and again

it was tightened up very slowly and the teeth carried as much farther apart as could reasonably be done. The gutta-percha filling was warmed slightly with a burnisher and again packed hard against the bicuspid. The patient was again dismissed for a few days. This was repeated to the fourth time, when the separation was sufficient, the tooth having been moved to its normal position.

Then the patient was instructed to do heavy chewing on that side of the mouth, in order to have the membranes of the teeth toned up to their full vigor by usage in mastication, as I intended to place a gold filling, and desired to have the peridental membrane in the best condition to withstand the malleting. The patient could now chew without any discomfort or injury to the gingiva from food wedging into the interproximal space.

Three weeks after the separation was last applied, the cavity was filled. The filling was made sufficiently broad mesiodistally, with a good contact. The gold which formed the contact was very thoroughly condensed, in order that it might withstand the wear which would occur from the bucco-lingual movement of the teeth in chewing. The separator was in position while the filling was placed and held the first molar hard against the second molar, thus securing the support of the second molar against the malleting. In finishing the filling, the separator was tightened a little more to facilitate the finishing of the contact point.

Subsequently the patient was able to use this side of the mouth normally in mastication and without the slightest discomfort. This is the best plan of which I know for the management of cases of this kind. It should be understood that the separation may be gained, the filling placed and finished in one sitting in practically all cases in which the teeth have not dropped together.

In the illustrations of the Perry separators, it will be noted that there is a set of six instruments, shown in Figure 223, which are made of different sizes to fit various positions about the mouth, while a "universal" separator, shown in Figures 219 and 220, has been more recently designed to take the place of the other six. This universal instrument has screws for adjusting the bucco-lingual width. It is larger than those of the regular set; it is also more difficult to adjust and is more in the way because of its bulkiness. I would strongly advise the use of the set of six in preference to the universal instrument.

In addition to the regular set of six, there is an additional set of six "specials," designed for unusually large teeth. These are needed only occasionally, but are desirable and serve to make one master of practically every situation.

Occasionally a case presents in which the crown of a tooth has been lost by caries and the teeth on either side have moved until they overlap the root between them. A very simple method of gaining separation in such cases is to substitute a pair of extra long separating screws in a Perry separator, so that the jaws may be applied to the teeth on either side of the root and move them both away from the root, holding the space gained at each application by the use of gutta-percha, as in the case mentioned above in which slow separation was employed.

CHRONIC SUPPURATIVE PERICEMENTITIS

ILLUSTRATIONS: FIGURES 226-264.

Thus far we have given our attention to the various common causes and forms of gingivitis. We have also noted the gradual destruction of all of the investing tissues of the teeth by deposits of salivary calculus. We are now to consider that condition in which, as a result of a preceding gingivitis, there occurs a detachment of the peridental membrane from the cementum, beginning at the gingival line, forming a pus pocket alongside the root. This is chronic suppurative pericementitis.

As previously mentioned, I applied the term phagedenic pericementitis to this condition in 1882. The word phagedenic is derived from the Greek phagein, which means to destroy by eating. In pathology this word was formerly much used to designate a suppurative condition in which the tissues were progressively destroyed; as a phagedenic ulcer. Phagedenic pericementitis, therefore, refers to that condition in which the peridental membrane is gradually destroyed by suppurative inflammation. However, the term chronic suppurative pericementitis describes the condition as accurately and is more readily understood. In ordinary usage the term *suppurative pericementitis* will be sufficient.

I am quite certain that this disease was destroying many dentures in ancient days, and has continued to do so to the present time, yet recognition of this fact has come in comparatively recent years. I was surprised on reviewing Dr. Rehwinkel's famous article before the American Dental Association in 1877, to find that he made no mention of such a form of disease. If he had known of these pus pockets it seems certain that he would have mentioned them. Neither is there any evidence that Dr. Riggs recognized this as a separate form of disease. It now seems probable that the descriptions of pus pockets which I wrote in 1882, and the figures illustrating them, published in the American System of Dentistry in 1886, constituted the first detailed description of them, and yet this is rather difficult for me to believe. I had recognized this form of disease in a vague way many years earlier, but did not have that mental grasp of

the condition as a whole, which enabled me to describe it fully. Many others no doubt had similar impressions. (See Figures 226-264.)

Up to the present time very few men have really studied the beginnings of the formation of pus pockets alongside the roots of teeth. The beginning of these is by many dentists still being attributed to deposits of salivary calculus. Many have also regarded the deposits of serumal calculus, on that part of the root which has been stripped of its membrane, as the principal cause of the continuance of the inflammatory movement. The fact seems to be that neither of these propositions is true. With the exception of the pus pockets caused primarily by serumal calculus in the subgingival space, which have been described, it is very rare that a pus pocket is caused by any form of calculus. In these pockets the deposit of calculus is a result of the inflammation and suppuration. It is derived from the serum escaping from the tissues in the inflamed and suppurating state — a result of the condition, not a cause of it. It seems safe to say that closer studies in the future will give these propositions general credence. They will be discussed more in detail later.

As a general proposition dentists have not, up to the present time, found or recognized those conditions leading to the formation of pus pockets. The treatment which has been given has been instituted after the disease has been fully under way, instead of at a time when it can be prevented, as it should be. The failure to connect the causes with the disease has been due largely to the great resistance offered by the tissues and the very slow progress of the disease. It is often many years from the beginning gingivitis until the case progresses to the point where there is an actual detachment of the peridental membrane. The gingivitis often causes little discomfort to the patient; there may be no complaint, and the dentist, whether he notices the inflamed area or not, does nothing to correct it. When the case has reached the serious stage of a definitely formed pus pocket, the original gingivitis has been forgotten.

In order to illustrate the progress of the disease, Figures 226 to 230 on the colored plate have been arranged to show the steps from the initial gingivitis to the formation of deep pockets and a lateral abscess. If one will imagine these to be progressive stages of a single case of many years duration, a better idea may be gained of the relationship of the gingivitis to the formation of the pus pocket.

FIGS. 226 TO 231. Reproductions of cases of gingivitis and pericementitis in various stages.

FIG. 226. It will be noticed that there is just a little inflammation of the septal gingiva between the first and second molars. This was due to leakage of food through a weak contact; there was no decay. It should be recognized that every such inflammation may be the forerunner of a case of chronic suppurative pericementitis, which may involve the entire denture. Here is the opportunity to *cure the gingivitis* and *prevent the pericementitis.*

FIG. 227. Occlusal view of a case similar to that shown in Figure 226. In this, food impaction occurred between the bicuspids. There is a little swelling of the tissue of both buccal and lingual embrasures with a depression between. A simple operation to restore a proper contact will result in the prompt disappearance of the gingivitis. If neglected it will progress to the condition shown in Figures 228, 229 and possibly 230.

FIG. 228. Buccal view of a case in which an open contact permitted food lodgment and resulted in the destruction of much of the septal tissue and detachment of the peridental membrane from the distal surface of the cuspid. This case has already progressed so far that a cure is out of the question. Most such cases will gradually progress with the best of treatment.

FIG. 229. A case in which the open contact has resulted in the complete destruction of the septal gingiva and the formation of a deep pocket between the first molar and second bicuspid. The trans-septal fibers have all been destroyed and there is little hope of maintaining a contact. The inflammation between these teeth will usually cause neighboring contacts to open, as described in the text.

FIG. 230. A lateral abscess, which occurred in a case similar to that shown in Figure 229. Instead of the pus escaping alongside the tooth, it penetrated the soft tissue and an abscess developed. The swelling caused the soft tissue to fill the septal space. After the acute symptoms have passed, the tissue of this space will resemble that shown in Figure 263.

FIG. 231. A case in which an open contact between the upper central and lateral incisors had caused a recession of the septal gingiva. Such cases usually progress to the destruction of the peridental membrane, if the contact is not restored.

FIG. 226.

FIG. 227.

FIG. 228.

FIG. 229.

FIG. 230.

FIG. 231.

Fig. 232.

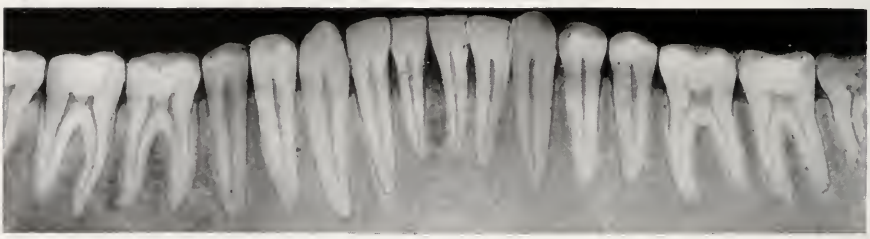

Fig. 233.

Figs. 232, 233. Panoramic radiographic view of a normal denture, to show especially the height of the bony alveolar septi between the teeth, for comparison with other radiographs in which destruction of bone has occurred.

These and the other panoramic radiographic illustrations in this book were prepared by Dr. Arthur D. Black. They are made by taking a full set of radiographs on the ordinary small films. From each of these a 4 by 6 inch enlargement was made. The enlarged photographs were cut and patched together, and then reproduced by the engraver.

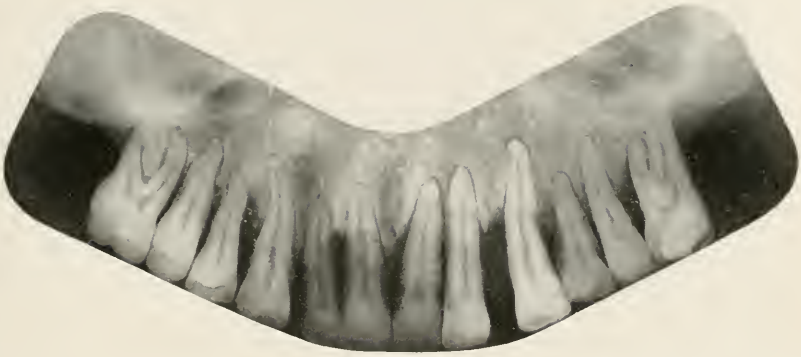

FIG. 234.

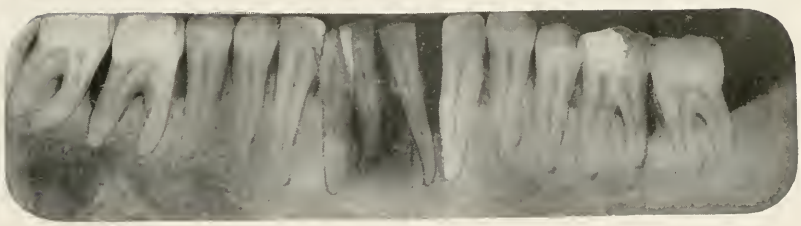

FIG. 235.

FIGS. 234, 235. Panoramic radiographic views of the upper and lower jaws in a case of chronic suppurative pericementitis of long standing. This patient had suffered from gout for five years. The right foot was first swollen and was very painful. In subsequent attacks the ankle was involved. The patient stated that except for this, he had never been sick a day. One lower incisor had become so loose that it was removed with the fingers. Pressure upon the gums caused pus to exude about the necks of many of the teeth. It was advised that all of the teeth be extracted.

FIG. 236.

FIG. 237.

FIG. 236. Normal peridental membrane. Section showing fibers attached to the cementum from the gingival line almost to the apex of the root. The section is not quite parallel with the long axis of the tooth and is a little to one side of the apex of the root. The fibers which pass upward into the gingivæ, those which pass to the crest of the alveolar process, and those which pass directly from the cementum to the bone are clearly shown as a practically solid mass from the gingival line to the end of the root. Photograph by Dr. F. B. Noyes.

FIG. 237. Normal peridental membrane. Higher magnification of about the gingival half of Figure 1. The fibers are shown more distinctly. The row of cementoblasts may be seen lying along the surface of the cementum. These cells occupy most of the space between the fibers as the latter enter the cementum. Photograph by Dr. F. B. Noyes.

Figures 236 to 240 were first published in an article by Dr. Arthur D. Black in the Western Dental Journal, Vol. 28, October, 1914, p. 1.

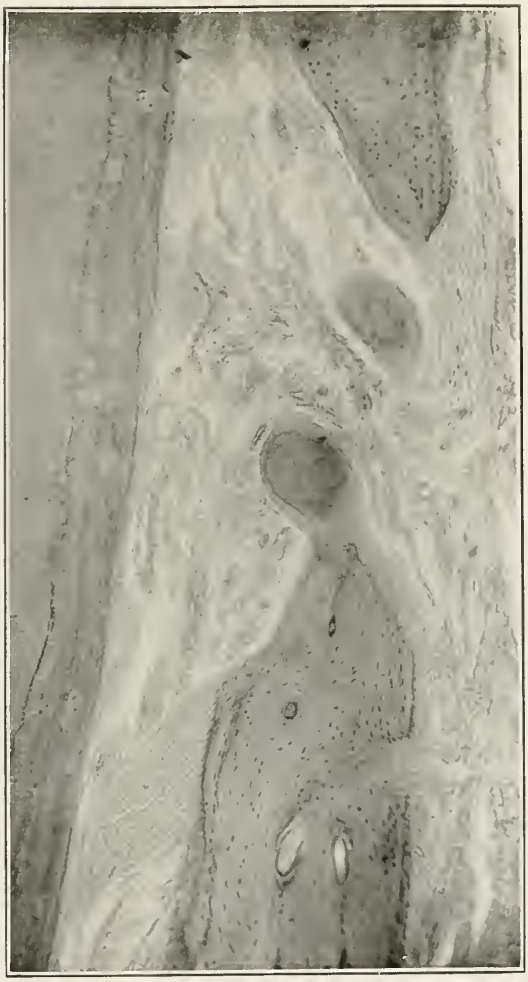

FIG. 238.

FIG. 238. Section through root and labial alveolar process of an upper central incisor, showing about one-fourth of the root close to the apex. Patient forty years of age. The peridental membrane on the lingual side of this tooth was detached almost to the apex and the pocket extended around on both the mesial and distal sides of the root, but was not so deep on either the mesial or distal, as on the lingual. This tooth was extracted by Dr. Arthur D. Black, May 27, 1912. With the patient under nitrous oxid anesthesia, two incisions were made through the labial gum parallel to the length of the root and a third incision was made horizontally above the position of the apex, meeting the other two. The alveolar process was cut through with a drill in the engine along the same lines, and the tooth with the labial peridental membrane, alveolar process and gum tissue were all removed together. This illustration should be compared with Figures 236 and 237. Marked changes have taken place in both the peridental membrane and alveolar process. Many of the fibers have disappeared. One strong bundle of fibers remains toward the apex of the root; another fairly good bundle is seen near the top of the illustration. Section prepared and photographed by Dr. F. B. Noyes.

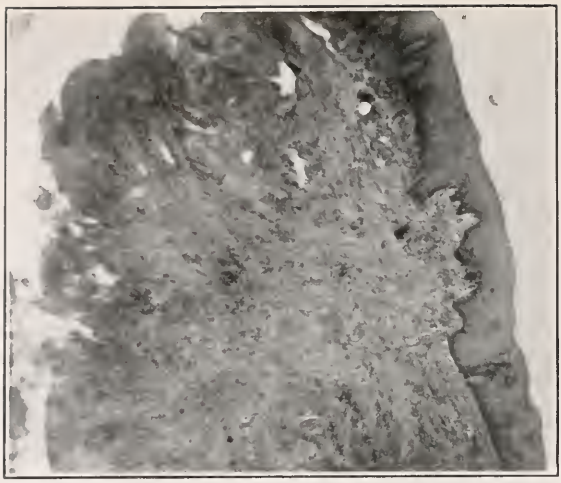

Fig. 239.

Fig. 240.

Fig. 239. Section through soft tissue overlying a deep pocket of many years standing on the labial side of the root of a lower left cuspid; from about the middle of the length of the root. Patient sixty-five years of age. Tissue cut away by Dr. Arthur D. Black on September 29, 1913. Normally the crest of the alveolar process should be present in a section cut in this position. The bone has all disappeared, as have practically all of the fibers of the peridental membrane. Section prepared by Dr. H. A. Potts, photographed by Dr. F. B. Noyes.

Fig. 240. High magnification from very near the center of section shown in Figure 239. The normal cells of the tissue have practically all disappeared and have been replaced by inflammatory tissue. By what process of treatment might we expect to have a regeneration of those elements necessary to a stable reattachment to the cementum? Section prepared by Dr. H. A. Potts, photographed by Dr. F. B. Noyes.

FIG. 241.

FIG. 241. Section through soft tissue overlying a pocket on the buccal side of the mesial root of a lower first molar, of possibly three to five years standing. Patient thirty-five years old. Tissue cut away by Dr. Arthur D. Black, August 3, 1914. The epithelial covering is shown at the top of the illustration, the lower part being toward the root. Bundles of fibers are seen, with much round cell infiltration. Specimen prepared by Dr. H. A. Potts. Photomicrograph by Dr. F. B. Noyes.

Fig. 242.

Fig. 242. Section through soft tissue overlying a pocket on the buccal side of the root of a lower first bicuspid, evidently of long standing. Patient fifty-five years old. Tissue cut away by Dr. Arthur D. Black, November 16, 1914. Although some bundle-like arrangement of fibers may be made out, this would hardly be recognized as having once been peridental membrane. Specimen prepared by Dr. H. A. Potts. Photomicrograph by Dr. F. B. Noyes.

Causes Leading to Formation of Pus Pockets.

Gingivitis always precedes. Some form of gingivitis always precedes the formation of a pus pocket. Deposits of serumal calculus in the subgingival space, or the various forms of injuries to the gingivæ which have been mentioned, are the most frequent forerunners. As has been stated, deposits of salivary calculus usually destroy all of the tissue overlying the root to a depth even with the deposit, and pockets alongside the root are not formed, as a rule, except in the later stages. While superficial suppuration of the tissue which is covered over by the deposit is commonly present, it is only in a limited number of cases that the peridental membrane is stripped away from the cementum in advance of the destruction of the overlying tissue. Deposits of salivary calculus should not, therefore, be considered as a cause of suppurative pericementitis in other than exceptional cases.

Deposits of serumal calculus on the enamel. Deposits of serumal calculus on the enamel within the subgingival space cause most of the pus pockets occurring on buccal, labial and lingual surfaces. As has been explained, the presence of these deposits causes or increases the inflammation of the gingivæ and suppuration occurs. Sooner or later the attachment of the peridental membrane to the cementum becomes involved and is cut loose. This is the beginning of a case of suppurative pericementitis.

Injuries to the gingivæ. By far the largest number of cases occur as a result of injuries to the gingivæ, and of these the injuries to the septal tissue by the impaction of food between the teeth in any of the many conditions cited, are both the most frequent and the most serious. In these cases, the irritation caused by the presence of the impacted food, and the poisoning of the tissue as a result of the decomposition of the food, both play a part in establishing and maintaining the inflammation. In some cases the impaction may of itself result in the beginning detachment of the peridental membrane at the gingival line. More frequently a suppuration occurring in the inflamed tissue causes the first detachment. Both may be more or less responsible.

Systemic conditions. Many men have believed that systemic conditions have been the principal factors in causing the diseases of the membranes about the teeth. I have already referred to this in the brief historical sketch of the development

of our knowledge of the diseases of these tissues. It must be recognized that the general physical condition of an individual will have its effect on the progress of such a lesion in the mouth, yet I can not to-day, from a review of the literature, or from my own investigations, believe that more than a very limited number of cases originate from systemic causes. I have gradually come more and more to the belief that careful observation and records during the early stages will establish the fact that there is a local exciting cause in nearly every case.

Specific infections. The theory that some particular organism is directly responsible for this chronic suppurative condition is a very natural one, and such an organism has been searched for almost continuously for many years. This has already been mentioned. On the basis of our present knowledge, it would seem that the organisms concerned in the destructive detachment of the membrane are factors by which the tissue becomes involved secondarily to some of the irritations mentioned.

Endameba buccalis. During the past year a number of interesting articles* have appeared relating the finding of endamebas in these pus pockets about the teeth. The first of these was by Dr. M. T. Barrett, of Philadelphia. There seems to be no question but that these organisms are present in practically all pockets about the teeth. It also seems that they are present secondary to some other primary exciting cause of the gingivitis. The study of the endamebas and their full relation to this disease has not yet progressed sufficiently to justify a full discussion, and therefore only a brief statement will be given.

Endameba is a term applied to parasitic ameba. The *endameba buccalis* is found in the depth of pus pockets about the teeth. Some of the contents of the pocket may be diluted with normal salt solution and microscopical examination will reveal

* Barrett, M. T.: The Protozoa of the Mouth in Relation to Pyorrhea Alveolaris, Dental Cosmos, Vol. 56, 1914, p. 948.

Smith, A. J., Middleton, W. S., and Barrett, M. T.: The Tonsils as a Habitat of Oral Endamebas, Journal American Medical Association, Nov. 14, 1914, p. 1746.

Bass, C. C., and Johns, F. M.: New Orleans Med. and Surg. Jour., Vol. 67, 1914, p. 456.

Barrett, M. T.: Clinical Report on Amœbic Pyorrhea, Dental Cosmos, Vol. 56, 1914, p. 1345.

Evans, J. S., and Middleton, W. S.: Endamebic Pyorrhea and Its Complications, Journal American Med. Assn., Jan. 30, 1915, p. 422.

Bass, C. C., and Johns, F. M.: Pyorrhea Dentalis and Alveolaris, Journal American Medical Association, Feb. 13, 1915, p. 553.

Endabemas in Pus Pockets About the Teeth. Editorial, Journal of the American Medical Association, Feb. 13, 1913, p. 593.

the presence of the motile endamebas. The following description is given by Bass and Johns in the February 13, 1915, issue of the Journal of the American Medical Association.

"They vary considerably in size and shape but with very little experience they are easily recognized. The clear ectosarc may project one or more pseudopodia, draw them back, project others in another direction, or the whole parasite may flow slowly in one direction or another. A pseudopod of ectosarc seems to always precede endosarc when it moves. Granules resembling portions of red blood cells in the endosarc help to give to the parasite an appearance similar to that of E. histolytica. The largest of the buccalis are about the size of the histolytica. Stained specimens are perhaps more easily diagnosed, especially by those not very familiar with endamebas. They may be stained by any one of several different methods, but the following is good enough for all ordinary purposes: (1) Spread pus on slide; (2) fix with heat; (3) pour on carbol fuchsin; (4) wash off at once; (5) stain with Loeffler's methylene blue one-fourth to one-half minute; (6) wash, dry and examine with oil-immersion lens. With this stain the endo and ectoplasm are well differentiated and the parasite can be differentiated from the much more numerous pus, epithelial, and other cells present. Their size varies from about three times the diameter of a pus cell down to smaller than pus cells. The large parasites contain in their endoplasm one or more large, dense, staining bodies or granules which appear to be the nuclei of pus cells. There is usually a clear zone around each of these, and also around the entire parasite. This is apparently due to shrinkage in drying or fixing the preparation. When stained with this stain the endamebas are darker stained than the pus and other cells in the preparation. In a preparation containing many parasites they can be seen with the low power lens studding the field as blue dots."

To examine for these organisms without staining a very fine pointed pipette should be introduced deeply into the pus pocket and a small quantity of its contents drawn into the pipette. In order to dilute it, three drops of steril salt solution should be put into a test-tube of one-fourth inch diameter; then the point of the pipette should be introduced into the salt solution and its contents forced out of the pipette into the salt solution. A glass slide with a depression should be used to receive the specimen to be examined. Around the depression a smear of vaseline should be placed and a cover-glass dropped over it.

The slide may then be placed in the thermostatically governed warmed stage. When the temperature reaches 98° the amebas, if present, become motile, and may be readily distinguished from the other organisms, tissue cells, etc. The use of an endamebacide in treatment is mentioned on page 199.

Symptoms and Tissue Changes.

The symptoms of suppurative pericementitis are those of the preceding gingivitis plus those which come with the gradual development of the pus pocket. A careful study of a large number of cases presenting these pockets beside the roots of teeth shows this condition very unique in character. No other condition like it can occur in the human body because there is no other such relation of tissues as exists here. If periosteum is stripped from bone, a portion of the bone dies, and is separated and exfoliated by the action of the deeper cells within the bone; when the cells which build the cementum are stripped from it, it also becomes a dead tissue, but there is no provision for its exfoliation, and it therefore remains as a constant irritant to the overlying tissue. This has been explained in the historical summary of the planting of teeth, including replanting, transplanting and implanting. In some of these cases, attachment of the soft tissues to the cementum occurs, but this attachment has been shown to be physiologically unstable, as manifested by the absorption of the planted root and the falling away of the tooth.

Locations of pus pockets.

The pus pocket is most frequent as an original or beginning disease in the biscuspid and molar region — the region of the mouth in which the heaviest work of mastication occurs. By far the greater number of cases occur from injury of the septal tissues by the impaction of food between some particular teeth in eating. For some reason the contact points between certain teeth begin to allow food, which is a bit tough and stringy, to pass and lodge between the two teeth, causing an inflammation of the septal tissue. In a similar way, a more limited number of cases occur in the incisor region. Cases which occur as a result of the deposit of serumal calculus on the enamel in the subgingival space, or of any of the injuries to the gingivæ which have been mentioned, may be observed about teeth in any position in the mouth. Deposits of serumal calculus in the subgingival space occur most frequently on the front teeth.

CHANGES IN THE TISSUES.

The steps in the destruction of the investing tissues in this disease are: (1) Gingivitis which affords the opportunity for infection. (2) Suppurative inflammation which detaches the peridental membrane from the cementum. (3) Destruction of the cementoblasts, gradual disappearance of the fibers of the peridental membrane of the detached area. (4) Absorption of the area of the alveolar process to which the detached fibers were connected. (5) A granular condition of the inner surface of the soft tissues covering the pus pocket. (6) Absorption by the denuded cementum of the products of the suppurative and putrefactive processes.

These changes may be recognized by a close observation of progressive cases, by the use of very thin flat explorers to determine the depth of pockets, by microscopical examination of sections of the tissues, and by radiographs taken at a favorable angle across a root which has a diseased membrane.

APPEARANCE OF THE GINGIVÆ. The condition of the gingivæ and overlying soft tissue varies remarkably. In some cases in which the pus pockets are very deep, the gingivæ look well. Sometimes they are only a little shortened. They present no distinct redness except in the paroxysms of acute inflammation. In other cases the soft tissues are almost constantly inflamed and red. The crest of the gingivæ overlying pus pockets often has a peculiar appearance which is more or less characteristic of the condition. The edge of the gingivæ may be rather smoothly rounded away from the surface of the enamel, and will have an unusually smooth, glossy appearance. This will include only the margin and possibly one or two millimeters of the outer surface. This strip may be very slightly bluish in color, in comparison with the adjacent tissue.

The following case is one in which I overlooked a deep pocket. A physician, with whom I had been intimately associated for a number of years, came in for examination, complaining of a lower first molar. His mouth appeared to be in excellent condition, except for a cavity in the mesial surface of the tooth of which he complained. I at once prepared the cavity and placed a gold filling. The next day he returned complaining that the tooth was very sore and extremely painful to the touch of the upper tooth. I then discovered what I had overlooked before, that the septal tissue was detached from the distal side of the distal root of this tooth. Taking a subgingival

explorer, I passed it alongside the distal surface of the first molar and it entered a pocket which extended almost to the apex of the root. So much tissue had been destroyed that it was necessary to extract the tooth. Had I been more careful in my examination on the previous day, I could have saved both the patient and myself the trouble of the gold-filling operation.

Infection and detachment. Subsequent to such injuries as have been mentioned, a pyogenic infection may occur and a little pus may be formed. At first this may be only the microbic decomposition of some plastic exudate thrown out by the inflammatory movement, and no actual tissue destruction may occur. The condition is that of constant reinfection, for there is no time that the saliva is free from micro-organisms which may produce pus, when the formation of plastic exudates gives them the opportunity. There may be pus formation in the tissue of the gingivæ apart from the attachment of the peridental membrane. Such an infection will heal at the first opportunity, which usually comes speedily, as would a slight pus formation in any other of the soft tissues.

At the next irritation and slight pus formation, the attachment of the peridental membrane at the gingival line may be involved, by cutting away some portion of the fibers from the cementum. This may be repeated and, little by little, fibers may be destroyed; or as a result of a more violent infection, a larger area of fibers may be detached.

According to the view that we can not obtain an attachment to the cementum except in aseptic conditions, there is no chance whatever for a reattachment. The suppuration may cease for a time and the soft tissue heal, except that this attachment to the cementum is not renewed. This occurs time after time, and the destruction goes deeper and deeper with each renewal of the inflammatory movement.

Pus may or may not be present at the time of examination. In many cases, on pressure upon the lateral sides of the arch with the fingers, pus wells up from under the margin of the gingivæ or about the tissue which remains. This pus is often undergoing putrefactive decomposition and is very foul smelling. In advanced cases, numerous pockets are constantly exuding pus and the products of decomposition, making the patient's mouth a veritable cesspool, and we can but marvel at nature's wonderful resistance which protects and prolongs the lives of so many persons whose mouths are in this condition. Every type of case will be seen, between this and the mildest appearing

cases in which no trace of pus may be found, although the subgingival explorer may reveal deep pockets.

Pockets progress most toward apex of root. The pockets gradually become deeper, often following one side of a root, or even a narrow space, but always progressing toward the apex of the root. In some instances a wide destruction of the peridental membrane over the side of a root may occur during a single inflammatory movement. But whether the destruction be rapid or slow, there is a constant tendency to destroy, progressively, toward the apex of the root, rather than to spread around the root. This fact has been fully established by a careful examination of cases; many of them at numerous intervals during their progress.

Some questions as to the exact pathological conditions and tendencies remain for future consideration. From all that I can now see, the peridental membrane itself is followed in preference to other tissues in this destructive process. This process may go on even to the apex of the root of a tooth, while the peridental membrane may remain healthful over other portions of the root. The remaining attached tissue may, and often does, sustain the tooth so completely that it performs the usual service in mastication. Finally a time comes when a tendency to spread around the root of the tooth occurs. This is usually marked by an increase in the amount of pus discharged and a rapid loosening of the tooth.

There is one point in the pathology of the peridental membrane that will be noticed as differing very materially when comparisons are made between disease beginning at the apex of the root and disease beginning in the gingivæ. There is no such disposition for the destructive effect in alveolar abscess to follow the peridental membrane toward the gingivæ, as in disease beginning in gingivæ to follow the peridental membrane toward the apex of the root. This marks the two affections as differing from each other, although both are suppurative. Why this difference I have found no satisfactory explanation.

In acute alveolar abscess, as ordinarily seen, there is no destruction or detachment of fibers of the peridental membrane from the end of the root. This occurs only in chronic cases. The destructive process is directed toward the bone surrounding the end of the root, and very soon the abscess is mostly within the cancellous portion of the bone. It does not follow the peridental membrane.

Failures of reattachment. Failures of reattachment of the

soft tissues to the cementum, which has been denuded, is a constant principal factor. I have carefully nursed comparatively small breaks in the attachment of the membrane upon the buccal or labial sides of the teeth, which I could plainly see, and have brought them back to a condition of apparent health. But this has been at the sacrifice of the length of the gingivæ. If I did not cut this away to uncover the pocket, it would shrink down after healing was accomplished, leaving a notch in the line of the crest of the free gingivæ. The healing was not from reattachment to the cementum, but by the tissues accepting a new line of gingival attachment. Even then, a depression is left which catches debris and is difficult to clean perfectly, and thus continues as a menace to the health of the parts. Such cases show very plainly the difficulties accruing as a result of the general failure of reattachment.

Cases tend to progress. When suppuration has, as a result of inflammation of the septal tissues, destroyed the attachment of some part of the peridental membrane to the cementum between two bicuspids, between a bicuspid and a molar, or between two molars, there is not much chance that it will cease. The infection is shut in completely by the two teeth, by the covering of the remaining septal tissue and by the lodgment of food debris. Fresh infection is constant under conditions favorable for its action. For these reasons the suppuration generally continues and goes deeper into the attachment of the membrane to the cementum.

Destruction of cementoblasts, fibers of peridental membrane, and the alveolar process. Subsequent to detachment from the cementum, the changes which take place in the investing tissues are of first importance in the consideration of these cases. These were fully presented in the discussion of the physiological functions of the various elements contained within the peridental membrane. It was pointed out that the fibers of the peridental membrane are cut off close to the cementum, that these fibers are subsequently absorbed and later the portion of the bone of the alveolar process to which they were attached also disappears by absorption. The cementoblasts are also destroyed over the area of detachment. There remains a tissue which, having lost those special elements which characterize the peridental membrane, has also lost the functions performed by those elements. The denuded cementum has become saturated with the elements of suppuration and decomposition, rendering it negatively chemotactic to those cells within the overlying tissues which

might otherwise attach themselves to it in the manner in which attachment may occur to planted teeth.

The disappearance of the cementoblasts and principal fibers of the peridental membrane is shown by microscopical examination of sections of the tissue. Such sections show the tissue to be more or less filled with inflammatory elements which have replaced the bundles of fibers and the cementoblasts. Depending upon the extent and duration of the pocket, there may remain scattering long fibers or none at all.

The series of illustrations, Figures 236 to 242, are presented for the particular purpose of showing the changes which occur in the tissues. Figures 236 and 237 are of normal peridental membrane, the fibers of which, together with the cementum and alveolar process, are beautifully shown. These may be compared with the other illustrations of the series, in which the extent to which the special elements have been lost can be observed. For example, Figures 239 and 240 are from tissue cut from a pocket, apparently of many years' standing, and there is nothing left in this tissue by which it could be identified as having ever been attached to the root of a tooth. The specialized elements have all disappeared. Figures 241 and 242 show some fibers remaining, but much infiltration of inflammatory elements. The three sections, Figures 239, 241 and 242, were all cut from positions in which there would have been bone of the alveolar process under normal conditions, but it is entirely gone. It would seem that these illustrations alone should be sufficient to establish the fact that a normal reattachment of the tissue to the root is impossible.

Absorption of alveolar process best shown by radiographs. The absorption of the alveolar process is best shown by radiographs, although in cases in which considerable progress has been made, the absence of the bone may be easily noted by digital examination. Oftentimes a rather sharp edge of the remaining alveolar process may be felt and the contour of the roots may be made out where they are covered only by soft tissue. The radiograph has, however, demonstrated very clearly that the absorption of the alveolar process begins much earlier and is often more general and more extensive than had formerly been recognized. It has become my rule in the study of these cases, as a basis for prognosis and treatment, to have radiographs made of the entire alveolar process, as these show in a very definite way, the extent to which the tissues have become involved. A series of such radiographs, taken at stated inter-

vals for individual cases, furnish an accurate record of the progress which the disease has made.

Radiographs may be taken of pockets on a proximal surface by holding the film close against the alveolar process on the lingual side, and so directing the rays that they will pass through the interproximal space in the bucco-lingual direction. The absorption begins on the inner surface of the alveolar process, and as the case progresses the area of absorption becomes funnel-shaped, dipping at an angle toward the apex of the root. Also a certain amount of the peridental membrane farther apically is shown to be involved in suppuration, appearing as a fine line in the radiograph. This shows that the progress of the suppuration is practically confined to the peridental membrane. The absorption occurs secondarily, in part as a result of the detachment of the peridental membrane from the cementum and in part directly by the suppurative process. I am inclined to the belief that the detachment of the peridental membrane from the cementum is the principal factor in causing the loss of the bone.

Many radiographs, made by the most experienced radiographers, show the extent to which the bone has been destroyed on the labial, buccal or lingual surfaces, although it can not be determined from the examination of the radiograph alone whether the bone which has been lost is on the buccal or the lingual side of a root. This must be done by an examination of the tissues. If bone has been destroyed on both the buccal and lingual surfaces, this will generally be shown by the radiograph.

The accompanying illustrations, made from radiographs, present every stage of progress of absorption of the alveolar process. Particular attention is called to the panoramic reproductions of radiographs of entire dentures which have been prepared by Dr. Arthur D. Black. These give a wonderfully clear idea of the extensive destruction of bone which occurs in advanced cases. Figures 232 and 233 are of a normal healthy mouth of a young man who has been free from diseases of the teeth and investing tissues. The position of the crests of the septi of the alveolar process will serve as a basis for judging the extent to which the bone has been destroyed in cases represented by the numerous other illustrations.

Figures 234 and 235 are panoramic reproductions from a mouth in which there were but two normal septi — between the lower cuspid and first bicuspid on one side, and between the lower bicuspids on the other. Figure 261 is another panoramic

FIG. 243.

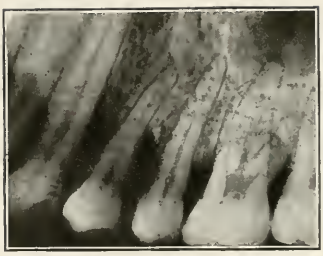

FIG. 244.

FIG. 245.

FIGS. 243, 244, 245. These three reproductions of radiographs are from the mouth of the same patient, who first consulted a rhinologist because of a suppuration of the maxillary sinus. In this case the gums and gingivæ made a fine appearance, notwithstanding the fact that several pockets were very deep. The separation of the teeth may be noticed in Figures 244 and 245.

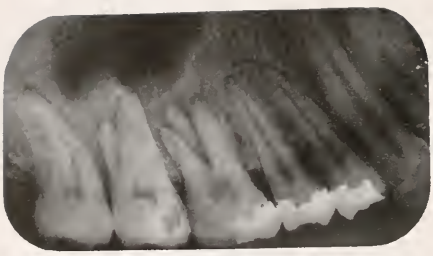

FIG. 246.

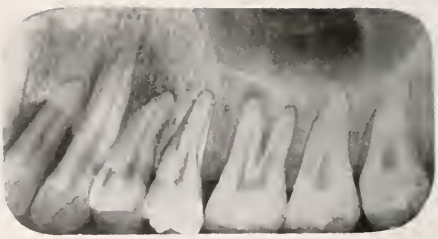

FIG. 247.

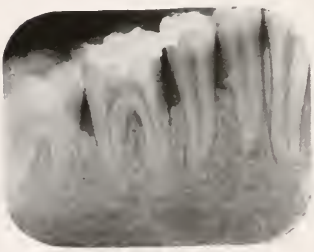

FIG. 248.

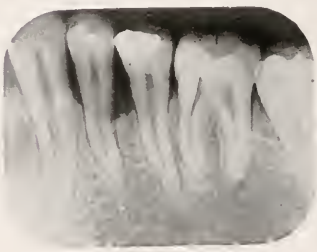

FIG. 249.

FIGS. 246, 247, 248, 249. These four radiographic reproductions are from the mouth of the same patient. In this case the gingivæ were much inflamed and pus could be pressed out about many of the teeth, although most of the pockets were not deep.

FIG. 250.

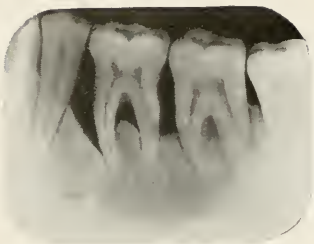

FIG. 251.

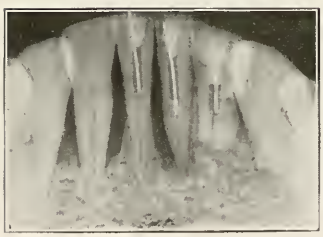

FIG. 252.

FIG. 250. The upper front teeth and remaining alveolar process, man forty-five years old. Pus could be pressed out about these teeth, particularly to the lingual. This patient had suffered from several attacks of arthritis.

FIGS. 251 AND 252. Reproduction of radiographs showing the funnel-shaped destruction of the bone of the alveolar process. The radiograph shown in Figure 252 was furnished by Dr. Thomas L. Gilmer. Small wires had been carried into the canals as far as possible just before the radiograph was made.

FIG. 253. FIG. 254. FIG. 255.

FIG. 256. FIG. 257. FIG. 258.

FIGS. 253 TO 258. Photographs of teeth with heavy deposits of serumal calculus. The membrane had been detached quite to the end of the root in Figures 253, 255 and 257. Figure 254 is a third molar which was only partially erupted, and the deposit covers almost the entire crown. Figures 256 and 258 are of cases in which the pockets were not very deep, but the deposits were heavy. All of the specimens are from Northwestern University Dental Museum.

Fig. 259.

Fig. 259. Ground section of a nodule of serumal calculus on the cementum. A little of the enamel is shown in the upper right corner of the picture. This illustration gives a good idea of the nodular forms generally found in pus pockets. I have often called this *pus pocket calculus.*

FIG. 260.

FIG. 261.

FIG. 260. Two views of a plaster model of a case of chronic suppurative pericementitis, showing the protrusion of the upper incisors, also the movement of the teeth. It required a special effort for this patient to hide the teeth with her lips. The separation of the central incisors followed the complete detachment of the transseptal fibers.

FIG. 261. Panoramic radiographic view of the upper teeth and alveolar process of the same case as Figure 260. This is about as great a destruction of the bone as can occur while the teeth remain in the mouth. Many nodules of serumal calculus may be seen on the roots. This patient, a woman of forty years, had suffered from a mild arthritis of many joints for more than five years.

Fig. 262.

Fig. 263.

Fig. 262. A lateral abscess between the second bicuspid and first molar. There was a deep pocket to the distal of the bicuspid root and the pus had penetrated the soft tissue instead of discharging alongside the tooth. These are often mistaken for true alveolar abscesses.

Fig. 263. Model showing the tissue destroyed by a septal abscess, practically the same as the case mentioned above, except that the septal tissue was principally involved. Many of these abscesses destroy quite a little bone of the alveolar process.

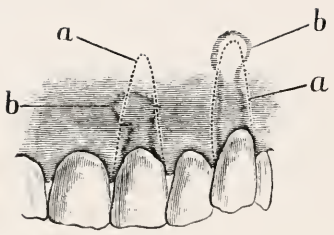

Fig. 264.

Fig. 264. Reproduction of illustration drawn by the author for the American System of Dentistry, 1886. (Vol. 1, Fig. 520.) This was probably the first illustration made of a pus pocket. The dotted lines a, a, represent the outlines of the roots of the teeth. The shaded lines, b, b, represent the extent of the detachment of the peridental membrane and destruction of the alveolar process.

reproduction of the process of an upper jaw showing about as extensive destruction of bone as can occur while the teeth remain in the mouth. Figure 289 is a similar panoramic view. Many other illustrations, from Figures 243 to 252, also Figures 287 and 288, show the destruction of the alveolar process. Figures 234, 235, 243, 244, 245, 251, 252, 261, 287 and 289 all show more or less of the funnel forms in the bone.

Granular condition of soft tissue covering the root. The inner surface of the soft tissue covering the portion of the root from which the peridental membrane has been stripped is in a constant state of inflammation, even though the superficial appearance of the tissue may be good. This tissue will be slightly or much inflamed at different periods, depending upon the infection present at the time and the condition of the root. The root may be smooth, without deposits, or there may be various forms of deposit from the flattest scales to the more irregular nodules.

Absorption by the denuded cementum of products of suppuration and putrefaction. The cementum is so porous that, in cases in which an infection has been in progress for a time, it will become saturated with the products of suppuration. The pockets are often invaded by putrefactive organisms which cause decomposition of material within the pockets and the cementum absorbs this also. This condition contributes to both the chronicity of the infection and the inflammation of the overlying soft tissue.

The porosity of both dentin and cementum was well illustrated in a case which came under my observation a few years ago. An upper central incisor in the mouth of a man forty years of age, was under treatment on account of the death of the pulp. The pulp had remained dead in the tooth for several years, causing it to be considerably discolored. After the treatment of the root canal had been completed and the apex of the root securely sealed with gutta-percha, it was determined to make an attempt to bleach the tooth, and twenty-five per cent pyrozone was sealed in for the purpose. There was no injury or inflammation of the peridental membrane. The pyrozone was sealed in at nine o'clock in the morning and by six o'clock the same evening the entire peridental membrane of the tooth was in a high degree of inflammation as a result of the penetration of the dentin and cementum by the pyrozone. The slightest touch upon the crown of the tooth caused sharp pain. By the next day the inflammation had subsided. This operation was

repeated, with similar results on two other days within the next two weeks.

Complaint of pain.

The complaint of pain in connection with these cases is very variable. The wide difference in the sensitiveness of the septal tissue to food impactions has been mentioned. As the depth of pockets increases, patients are more likely to complain of soreness of the teeth to the stress of mastication, than of pain. Cases may progress to the stage where many teeth are hopelessly involved without the least pain; on the other hand, there will be acute pain in many cases during a period of acute inflammation. Both the tenderness to touch and the pain are more apt to be complained of when the inflammation has involved the deeper tissues, near the apex of the root, and the swelling has caused the tooth to be slightly lifted in its socket. The opposing teeth strike it every time the mouth is closed, and this materially increases the inflammation, often causing the tooth to become extremely painful and tender to touch. If the tooth is given rest for a few days by some plan which relieves the occlusion on it, the inflammation will generally subside, and the tooth may again be used in mastication without discomfort. Some slight injury or a new infection may cause a repetition of the acute symptoms. This may occur again and again until finally in desperation the patient will consent to, or possibly insist upon, the extraction of the tooth.

Deposits of serumal calculus.

Deposits of serumal calculus may or may not be present within the pockets on the cementum from which the peridental membrane has been detached, although they generally are present. The deposit necessarily occurs subsequent to the detachment. The calcific elements are brought to the pocket with the serum which is exuded into the space from the overlying tissue as a result of the inflammation, in the same manner as deposits are laid down upon the enamel of the subgingival spaces. The nature of the deposit is the same; the only difference is in the form.

Deposit often nodular. The deposit of serumal calculus occurring in the subgingival space is, as has been mentioned, usually in the form of a flattened scale, due to the pressure of the gingivæ against the deposit while it is soft. Within the depth of a pocket, alongside the root, the soft tissue is not so

inclined to hug the root closely, and accretions to an original nidus of deposit are likely to be gradually built on and around, forming a nodule. Either the scale or nodular form may be present, although the deposit within the pocket is generally more or less uneven. The difference in the form of the deposit in the subgingival space and in the pocket is so marked that I have frequently used the term *pus pocket calculus* to designate the latter form. (See Figures 253 to 258, also Figure 261, and the series of illustrations, Figures 268 to 284.) Figure 259 is a microscopic section of a nodule attached to the cementum.

Occurrence of deposits. Cases are observed in which there are no deposits whatever in pockets of many years' standing. My recent studies of the nature of the deposit of calculus leads to the belief that for such individuals the balance between the quantity of food digested and the elimination is such that no excess of calco-globulin is present in the body fluids and therefore none is brought to these pockets with the serum which is constantly poured out into them. With this view, all such persons should also be free from deposits of salivary calculus, because if deposits of salivary calculus are occurring in a mouth in which there are pus pockets, we should expect a corresponding proportion of calco-globulin in the serum of the pockets. It does not necessarily follow, however, that salivary calculus will be present in mouths in which serumal deposits are present, as the care of the mouth may be sufficiently good to prevent the accumulation of deposits of salivary calculus, or the forms of the gingivæ may be such that no deposits occur, even though calco-globulin is present in the saliva. Careful clinical observations agree with these statements.

Deposit contributes to progress. When deposits have occurred within these pockets, they serve as an additional irritant to the overlying soft tissue, and tend to maintain the inflammatory movement. This irritation results in the continued outpouring of excessive amounts of serum, which will in turn bring additional calco-globulin in the proportion that it is present in the body fluids. The tissue is in such condition as to invite suppurative infections, and these occur frequently or continue with slight or no interruption. Thus it will be seen that while the deposits are secondary to pocket formation, they become one of the factors to the continued progress of the disease.

As mentioned in the consideration of gingivitis caused by deposits of serumal calculus, these deposits on the enamel may be the original beginning of cases of suppurative pericementitis,

though they account for but a small percentage. The first inflammation of the gingivæ may be due to the deposits of serumal calculus on the enamel in the subgingival spaces. This may provide a focus for a pyogenic infection which destroys a little of the attachment of the peridental membrane at the gingival line. Deposits then in turn occur on the denuded cementum. Another suppuration occurs, more tissue is detached, new deposits occur, and so the case progresses.

Enlargement of cervical glands.

During the progress of this disease there is apt to be swelling of the cervical glands, which drain the infected area in the mouth. It has often seemed to me that the amount of swelling of these glands was out of proportion to the inflammation about the teeth. In some cases, in which there is very slight inflammation about the teeth, there may be considerable enlargement of the glands of the neck, while in others, in which the mouth infection is extensive, none of the cervical glands can be palpated. The swelling of these glands should be looked upon as a danger signal, as it often indicates the beginning of a general infection which may do great damage by the establishment of foci elsewhere in the body. We must not conclude, however, that the absence of enlarged cervical glands indicates that there is no systemic danger, because most of the secondary infections are probably hematogenous. The submaxillary gland does not often partake in such inflammations, but it must be differentiated from the cervical glands.

Palpation of submaxillary and cervical lymphatic glands.

The most effective method of palpating the submaxillary gland is to place a finger of one hand in the mouth and pass it under the tongue to the lower border of the inferior maxillary bone, at about the position of the molar teeth, and a finger of the other hand on the skin below the jaw, grasping the gland between the two fingers. The two first fingers are best to use in this examination, as they are more sensitive to touch than the others. The first finger of the right hand should be placed inside the mouth in palpating the patient's right submaxillary gland, while the first finger of the left hand will be best in the mouth in palpating the patient's left submaxillary gland. Sometimes the little finger may be used instead of the first finger inside the mouth. The posterior end of the gland can usually be definitely made out, about even with the distal surface of the second molar. The anterior end is not so easily determined. The gland is

usually about the diameter of a finger, but varies considerably. If there is tenderness or much enlargement it may be discovered by such an examination.

Under normal conditions, the cervical lymphatics can not be palpated. When enlarged, they may be felt as movable nodules of variable size from that of a pea to a large filbert. The patient's head should be tipped forward to relax the muscles of the neck, which will make it easier to palpate these glands. If enlarged, they will usually be found just under the skin of the neck, a little below the lower border of the inferior maxillary bone and anterior to the sterno-clido-mastoid muscle.

Excitation of salivary glands.

There is an almost continuous excitation of the salivary glands of persons afflicted with this disease. I have often watched them carefully while sitting at rest in my chair, or in my reception-room, and counted the acts of deglutition. Often this will occur three to five times per minute. When questioned about drooling of saliva at night, many have told me that it was necessary to use a napkin to protect the pillow from this flow. Those who sleep lying on the back, swallow this material unconsciously during their sleep.

Movements of the teeth as a result of pocket formation.

One of the very important items in the study of the pathology of these cases is the movement of the teeth which occurs as a result of the swelling and the disturbance of the balance of pull of the various groups of fibers of the peridental membrane. In order to understand these movements, there are several principles which must be considered.

It is important that the general structure of the gingival tissues and the influence of the different groups of fibers which have been described, be held strictly in mind. The fact that these fibers *pull* one group against another in holding the teeth in the line of the arch, and that in normal conditions these *pulls* are accurately balanced, is an important matter, for in this field of pathology much havoc results from the disturbance of the balance of these *pulls*.

In this it is a general principle that wherever the fibers of the peridental membrane are being destroyed along one side of the root of a tooth, so that their normal pull upon the tooth is broken or much weakened, the *pull of the fibers upon the well side will draw the tooth away from the diseased side*, or tend to

do so. This is true wherever such a condition occurs and it will be effective unless there is some counteracting force to prevent such a movement. Sometimes deeply interlocking cusps with the teeth of the opposing arch will prevent such a movement, and many other conditions may prove temporarily sufficient, but in the long run all of these tend to give way.

In these cases there seems to be a weakening of the pull of the fibers in excess of the actual destruction of tissue. We know this most certainly from the cessation of the normal pull of the fibers during an inflammation which does not destroy them, and from which they recover and again become normal. This is seen most often in irritations of the septal gingivæ from food having been crowded between the teeth. Here the pull of the fibers may be so reduced that the teeth will actually stand apart after the food has been removed. But by care, the fibers will recover their tone and the closeness of the contact will be restored. This has been made use of extensively in the slow wedging process in filling teeth. The constant pressure by the wedge causes the fibers to relax, and they permit the teeth to be separated a considerable distance. After the wedge has been removed, however, the fibers recover their tone, and draw the teeth back into close contact.

Labial movement of upper incisors. When deposits of serumal calculus on the lingual surfaces of the upper incisors cause the detachment of a portion of the peridental membrane and the formation of pus pockets, the *pull* of the fibers in the lingual direction is weakened, and the tendency is for the *pull* of the fibers of the labial side to move these teeth labially, so that they begin to perceptibly protrude. At first this movement is so slow that the patient will not notice it, and also the disease progresses so slowly and painlessly as to pass unobserved. But after considerable time the patient's friends, it may be, will first call attention to the increasing prominence of the incisor teeth. Then the patient in some alarm may consult his dentist. Unless a very critical examination is made the cause of the movement may not be discovered, for in most of these cases the gingivæ on the lingual side give a fair appearance. The tissue is generally thick and heavy at this point, and especially well adapted to conceal the disease going on beneath it. It therefore often happens that the cause is overlooked. If, however, a subgingival explorer is used to examine the attachment of the membranes of these teeth at the lingual sides of the roots, deep pockets will be found with a slow suppuration in progress, which

is cutting away the attachment of the fibers more and more, weakening or actually destroying the pull of the fibers in the lingual direction. Therefore, in the cases in which considerable destruction of the attachment of the peridental membrane has occurred to the lingual of the incisor teeth, an amelioration of the conditions may occur as a result of careful treatment, but there will generally not be a stoppage in the movement of the teeth to the labial, because the harmony of pull has not been and can not be restored.

As this condition goes on from bad to worse, the forward movement of the incisors increases, and a time comes when the trans-septal fibers, which normally hold the contacts of the teeth solidly together, become involved in the inflammation and permit loosening of the contacts between the anterior teeth and later those in the bicuspid and molar regions, progressively backward.

This loosening of the contacts gives the opportunity for injury to the septal tissue of those regions. This leads to suppuration after suppuration, which goes on destroying the membranes of the teeth. The movements of the upper teeth bring about movements of the lower teeth, resulting in the formation of pus pockets between them. Finally suppuration is present generally in the peridental membranes and the whole denture becomes a wreck. Figure 260 reproduces two views of a plaster model of a case in which both extensive labial movement and wide separation has occurred. The panoramic view (Figure 261) is of the same mouth.

TEETH MAY MOVE FORWARD OF NORMAL POSITION OF LABIAL PROCESS. We should return now and examine the forward movement of the incisors in some of the features not previously noticed. The question may be asked: How is it possible for the incisors to move so far forward and do all of this pulling of the other teeth with them? It seems that the roots of these teeth actually pass the outer labial line of the alveolar process as it stood when normal. This is certainly true in many cases which I have had under observation.

This is accomplished slowly, step by step, by absorption from the labial alveolar process on its inner side next to the peridental membrane, and the building of bone on its labial side. In this way there is an actual movement of the alveolar process forward which carries the teeth bodily with it. While this is in progress the fibers of the peridental membrane on the labial side of the root of the tooth are being loosened from the alveolar

process in space after space and replanted farther and farther forward. In this way their pull is kept up while the teeth move labially, until it is with difficulty that the lips can be made to cover them. In closing the mouth, the teeth may overlap the lower lip. In this movement the crowns of the teeth move much more than the ends of the roots. In some cases I have seen the incisors in almost a horizontal position. A movement of the incisal edges of the teeth of a quarter of an inch forward is no exaggeration of what actually occurs. Such a movement of the teeth completely changes the expression of the face and it is often very difficult to bring the face into correct form with artificial teeth because the residual alveolar ridge has moved too far forward. Shakespeare seems to have known something of this change of expression, for in one of his plays he says of a woman acting a part: "She is not young, for her upper teeth are already becoming prominent."

In this there is a hint of the slowness of this movement of the teeth. For this process to run twenty-five years is no exaggeration of statement. It is so slow that few men have observed individual cases from the beginning to the end. It is largely for this reason that we have no descriptions, such as I have given, in our printed records. Especially, dentists have not followed cases with written records which make the facts clear. While I did not keep records of these cases in as much detail as a few men may be keeping them to-day, my records were sufficient to enable me to review the general progress of cases from their beginning until the teeth were finally lost. There is not a record of the cure of any one of these cases in which the labial movement of the incisors had become established.

These are among the most hopeless cases. The movement of the teeth is continuously progressive. As mentioned in the treatment of these cases, the inquiry has come many times as to the possibility of moving these teeth back into place and keeping them there. I have generally advised that they be extracted. If this is done before the separations in the bicuspid region have begun, the loss of these teeth may be averted. The attempt to retain the incisors too long has often been disastrous.

Multiple pocket formation. It has been observed that when a pocket has formed on the proximal side of the root of a tooth, or of the two teeth on either side of an interproximal space, and has cut away the trans-septal group of fibers uniting the two teeth, it frequently happens that other similar pockets

soon begin to form on the proximal sides of the roots of neighboring teeth.

Suppose, because of a faulty contact, there is an injury to the septal tissue between the first molar and the second bicuspid which destroys the trans-septal fibers. Very soon a similar injury may be noted between the first and second molars. If the case has been watched closely enough it will be found that the rule mentioned relative to the labial movement of the upper incisors because of pockets on the lingual sides of their roots, is followed. That is, there is a tendency in all cases of pocket formation for the teeth to move away from the diseased side during inflammatory periods. When this occurs, because of the diseased membranes of the proximal surfaces of the molars, the movement is more difficult because it is in the line of the arch instead of to the side of the arch. The first molar will be forced hard against the second molar, and if the stress is sufficient, the second molar will be moved distally very slightly. With the abatement of the inflammatory movement, this stress will be relieved, and the teeth will return to their former positions. This movement of the teeth will be repeated with each recurrence of the inflammation, and will often cause some slight interference with the intercusping of the teeth with those of the opposite jaw, which tends to make some movement of the teeth at every closure of the bite. This has its disturbing effect and is particularly liable to be such as to drive the first molar back into its former position and food may be forced between it and the second molar, thus establishing an inflammation of the septal tissue in this space. The movement of the second bicuspid mesially exerts a similar pressure against the first bicuspid, and by the same process food will be forced past the contact into that septal space also. As these movements continue there comes a loosening of the contacts of the neighboring teeth, and generally through the arch, by interference with the fibers which form the trans-septal groups, the function of which is to hold the teeth solidly against each other in the line. It seems to make little difference what causes the inflammation of the septal tissue first involved.

These inflammatory movements, occurring in the septal tissues, cause these groups of fibers to lose their tonicity. This is seen also in the fibers of the peridental membrane in acute apical pericementitis occurring as the beginning of alveolar abscess. The tooth often becomes very loose over night. This loss of tone is, indeed, the common effect of the involvement of

near-by tissues in inflammation. The result is a series of inflammations affecting a number of teeth, or the septal tissues generally.

When I first noticed the tendency in this disease to spread from one septal space to another, my thought was directed to some special infection as the cause. I studied this very thoroughly for a number of years. Many times I felt assured that the supposition was correct, but in the long run of observation the proof failed. The study of the movements of the teeth in local inflammatory conditions and the disturbance of the contacts which occurs, together with the increased freedom of movement of the teeth as the disease progresses, and finding of lodgments in neighboring septal spaces, furnishes very convincing proof that the spread of the disease from contact to contact is as described above.

The dentist who has a considerable number of patients under observation, who present the beginnings of this disease, having but one or two septal spaces involved, and will carefully examine neighboring contacts from time to time, as to their comparative tightness, studying the liability of food being forced onto the septal tissue, and frequently finding that food has been forced through the contacts, will soon become convinced that this purely mechanical cause of the spread of disease from one interproximal space to another is explained by the results. For the present, at least, it seems best to rest the case on this proposition.

Open contacts resulting from movements of teeth may be observed in many of the illustrations. Figures 234, 235, 244, 245, 260 and 261 all show separations which appear to have been brought about as described above.

Gingival abscess, septal abscess and lateral alveolar abscess.

An acute abscess occasionally develops in the investing tissues of the teeth in connection with the progress of these ordinarily chronic suppurations, and these abscesses are named according to the tissue in which each occurs. If for any reason the pus, which is formed in the depth of a pocket, is prevented from escaping alongside the root and into the mouth between the crown of the tooth and the margin of the gingiva, it may invade the overlying soft tissue and develop within a few hours an abscess with all the symptoms of an acute suppuration. If the pus pocket is on the labial, buccal or lingual side of a root and is not deep, the abscess will tend to point in the overlying

gingivæ, and this is termed a *gingival abscess*. If the pocket is on the proximal surface of a root, the septal tissue will be principally involved, and the condition is designated as a *septal abscess*. If the pus pocket is deep, the abscess will tend to point on the gum over the edge of what remains of the alveolar process, or may penetrate the process. This is a *lateral alveolar abscess*. Figure 262 is from a plaster model of a case which presented with a lateral abscess to the buccal of the upper second bicuspid. Several drops of pus were obtained when the swelling was lanced. The pulps were vital in all of the teeth shown. Figure 263 illustrates the destruction of tissue by a septal abscess. Figure 230 is also of a case of lateral abscess.

A case was brought by a dentist to the School Clinic for diagnosis. The symptoms were extreme soreness of the upper second bicuspid and first molar and swelling and inflammation of the tissues about the teeth, with much pain. This had been supposed to indicate acute alveolar abscess. There was no caries. The dentist had cut into the first molar, expecting to find a dead pulp, but found the pulp alive. He had then cut into the second bicuspid and found its pulp alive also. Then he cut into the second molar, and finally into the first bicuspid, and found the pulps in these teeth were alive. He then brought the case to me. I found the septal tissue between the second bicuspid and first molar very much swollen, with a rather widespread inflammation. The proximal surfaces of these two teeth stood apart. Taking a very sharp narrow blade, I pressed the handle far back into the cheek of the opposite side of the mouth, from which position I entered the point of the blade into the swollen septal tissue between the second bicuspid and first molar from the lingual side. Pus welled out around the blade. This was a septal abscess, the original cause of which was an open contact which permitted stringy food to pass. Two days later, when soreness had abated, I found some food debris still lying deep in the space beside the first molar.

Differential diagnosis from true alveolar abscess. As in the above case, such abscesses, and more especially lateral alveolar abscesses, are sometimes mistaken for true alveolar abscesses. In making a differential diagnosis, an examination should be made to determine whether or not the pulps of the teeth are alive. If so, this excludes true alveolar abscess. A peridental membrane explorer should be entered between the gingiva and the crown and passed alongside the root until it passes into the depth of the pocket. If the tissues are thus

shown to have been detached to a depth corresponding to the position of the abscess, the diagnosis is clear. As such an examination is often very painful, it may be best to use the lance first, as this is indicated anyway. In the literature these lateral and septal abscesses have been spoken of a number of times as the beginning of disease of the peridental membrane, evidently in cases in which the previously formed pus pockets have been overlooked.

Another condition of extreme soreness occurs frequently in the latter part of the progress of this disease, when the peridental membrane is being stripped from the apex of the root, or when the apex of the root is very closely approached. In these cases the symptoms closely similate those of acute apical pericementitis, or the beginning of alveolar abscess, and if the pulp of the tooth is alive, it generally dies as a result of the extension of the suppurative process. In all cases in which there are deep pockets, with or without the formation of abscesses, it is important that the condition of the pulp be ascertained.

Admixtures of suppurative pericementitis and inflammations caused by deposits of salivary calculus.

Thus far I have spoken of chronic suppurative pericementitis and the destructive inflammation caused by deposits of salivary calculus as entirely distinct conditions. This has been done to impress the important differences which exist. In most cases the distinction between the two is sufficiently clear, but in some it is not. Deposits of salivary calculus occur frequently during the progress of suppurative pericementitis. Such deposits may occur either before the formation of, or during the progress of the pus pocket without being in any way connected with it.

The appearance of the gingivæ in the two conditions is usually very different. In suppurative pericementitis there is not necessarily any salivary calculus present. We may say that generally there is none, if the cases are seen early. Yet the presence of some calculus is not necessarily excluded. When salivary calculus is causing inflammation of the gingivæ, the presence of the calculus is very apparent, and the reddening of the gingivæ is usually pronounced over a considerable area, or including from two or more teeth to a large part of the free gingivæ. In this the septal gingivæ are most generally not included until considerable progress has been made. Salivary calculus does not generally lead to the formation of pus pockets

during its progress. Its general tendency is to destroy the margin of the free gingivæ and shorten them as a first effect. This progresses slowly and finally invades and includes the septal tissue.

In the early stages of conditions leading to the formation of pus pockets, small points of inflammation are the rule, instead of the broader areas produced by salivary calculus. In the larger number of cases these will at first be confined to the septal tissue of some particular space, rather than to the free gingivæ. Usually the septal tissue begins to be shortened by the pressure of food into the space. This inflammation of the septal tissue generally continues for a long time before the beginning of actual injury to the peridental membrane.

As the disease of the gingivæ progresses and pus pockets form, the gingivæ are blunted; their borders, or crests, are thickened. Then if there is an admixture of calculus coming into the mouth with the saliva, it will be more liable to be deposited where the crests of the gingivæ are thickened than elsewhere. Occasionally cases present with heavy deposits of salivary calculus about teeth which have deep pockets. The examination of many cases will convince one that the deposit of salivary calculus is not responsible for the formation of the pus pocket. By removing the calculus and examining the peridental membrane with the subgingival explorer, one may get an understanding of the actual conditions. If there are no distinct pus pockets, the case is one of inflammation caused by salivary calculus alone. If, however, the gingivæ have been destroyed, and much shortened, and deep pus pockets are found, especially on proximal root surfaces, the case is one of chronic suppurative pericementitis, which has received deposits of salivary calculus later. The changes in the gingivæ, which have occurred with the progress of the pus pocket, have afforded the opportunity for the deposit of salivary calculus.

This statement may not be easily understood by those who have not studied closely the conditions controlling the deposit of salivary calculus after it comes into the mouth with the saliva. Any blunting or thickening of a gingiva as it lies against a tooth will present the opportunity for a deposit, while the same place would not collect calculus before the blunting or thickening of the gingiva occurred. The effect of this condition should have careful study.

Pus pockets may occasionally occur in connection with inflammations caused by deposits of salivary calculus, but the

pockets are generally much broader, reaching more around the root of the tooth. Indeed they differ so much from those occurring from the injuries mentioned that there should be little difficulty in diagnosis. They are not present in the beginning of salivary deposits, or at a time when treatments should be undertaken, but occur most in the later stages. Indeed in the later stages the two conditions come to look more and more alike. In the last stages of suppurative pericementitis we will often find considerable of the gum tissue standing up about the teeth, while in the final stages of the inflammations caused by salivary calculus there will be practically no gum tissue left.

TREATMENT OF CHRONIC SUPPURATIVE PERICEMENTITIS.

ILLUSTRATIONS: FIGURES 265-295.

In the treatment of the pus pocket, first consideration should be given to the relationship which such foci of infection bear to general systemic conditions. It has been sufficiently demonstrated that many remote lesions which often shorten the life of the individual are the result of these infected pockets. This being the case, the first rule of practice must be to protect our patients from this source of systemic poisoning. The question of the service which teeth with pockets may be giving in mastication should have no weight as against the general health. No set of natural teeth with many of these pockets about their roots is enough better in mastication than artificial teeth, to justify one in jeopardizing the health of the individual by their retention.

Our consideration of the general health should not be based on the apparent or even the actual physical condition of the particular patient at a given time, but rather on the danger to his future health. Many of the secondary systemic lesions are incurable when they have progressed sufficiently to be recognized. We are not justified therefore in permitting patients who are apparently well to go about with such foci of infection in their mouths. While we recognize the fact that the resistance of some individuals is sufficient to overcome these poisons for many years, possibly to old age, yet no one can be certain at any given time that such a person is really free from injury. Our highest duty in the management of these cases is to free our patients from the danger in which they are placed by such a chronic suppuration.

The key to the treatment of suppurative pericementitis is in the statement that suppurative detachments of the peridental membrane are permanent detachments. This fact should be constantly foremost in the mind of the dentist, as it naturally divides our consideration of the treatment into preventive, palliative and radical. Preventive treatment must be that which will in each case prevent or tend to prevent those conditions which result in suppurative detachments. Palliative treatment should

be such as will keep the tissues overlying detached areas in the best possible condition and thus retard further detachments. Radical treatment should be employed to cure this disease by root amputation or extraction.

Preventive treatment, which is by far the most important, consists of (a) the maintenance of the gingivæ in good health, and (b) the detection of inflammations and their cure before serious detachment has occurred. Palliative treatment is to be applied in those cases in which detachment has occurred, but in which conditions are such that the effort is to be made to keep the cases under control. Radical treatment by root amputation or extraction is indicated in cases in which it seems unwise to try or to continue palliative treatment.

Preventive Treatment.

All that has been said heretofore relative to the treatment of the various forms of gingivitis is essentially preventive treatment of suppurative pericementitis. The inflammations of the gingivæ, as such, are of little consequence and require treatment principally for the purpose of preventing involvements of the deeper tissues.

The plan for preventive treatment.

The treatment for prevention can be effective only as it is undertaken seriously and systematically. The plan must include: (1) The careful examination of the gingivæ as a part of the routine examination of each mouth; (2) a record of the areas of inflammation observed; (3) a study of the cause of each such area; (4) the treatment necessary to remove the cause; (5) the careful training of the patient in the care of the mouth; (6) subsequent examinations.

This service should come to be a considerable part of the practice of each dentist. It should be carried out along the same general lines which have already been discussed in the treatment of inflammations caused by deposits of salivary calculus. Those who are following this plan in the most thorough manner are finding that there is no end to the watchfulness required for most patients. In many mouths changes occur during each year which lead to new inflammations. Wear of proximal surfaces, wear of occlusal surfaces, slight movements of teeth, decays, accidents, etc., all tend to change conditions so that the mouth which is free from gingivitis at the close of a series of operations may, within six months, present several

new areas. It should be quite readily appreciated that mouths which are thus closely watched should not, and generally do not, develop pericementitis.

PREVENTIVE TREATMENT MUST BE BY GENERAL PRACTITIONER OF DENTISTRY. It will, I think, be realized that the treatment of the beginnings of disease of the gingivæ belongs to the general practice of dentistry in just the same way as the correction of the abuses of the gingivæ belongs there. The first element of this is the watchfulness of the practitioner for the beginning injury. In the very nature of things this must be discovered by the general dental practitioner, and not left to specialists. The rule has been that specialists do not see these cases early enough to apply preventive treatment.

It is the general practitioner who must discover deposits of salivary calculus, and he must so train and control his patients in the care of their mouths that these deposits will be prevented, if he will save the teeth from being lost from this cause. Likewise, the general dental practitioner is the one who must discover the deposits of serumal calculus on the enamel and remove them before serious suppuration occurs. The operations necessary for the prevention or cure of most of the injuries to the gingivæ are the routine dental operations, carefully and properly done. If one is practicing dentistry at all, he should be acute enough to see these things and skilful enough to prevent or correct them. Our greatest hope for the future is in the application of preventive treatment by all dentists, rather than in the use of palliative treatment by specialists.

SYSTEMATIC OBSERVATION AND INSTITUTION OF TREATMENT EARLY. Up to the present time but few dentists have applied treatment to prevent the formation of pus pockets, the large majority having deferred treatment until after the pockets have been formed. As a first effort in practice this must be exchanged for a systematic scheme of observation for the detection of the conditions which act in the causation of pus pockets, and the correction of these before they have done serious harm. We have become so inured to a certain phase of thought toward the soft tissue investments of the teeth — a phase of neglect of their appeals for help — that a change to greater care is more of a task than most persons would suppose. Somehow the treatment of dental caries by fillings and other operations has become the principal service of the dentist to such an extent that the soft tissues are neglected until it becomes apparent that

severe disease has developed, and that there is imminent danger that the teeth will be lost.

Care to avoid injury to soft tissues in all operations. It has been pointed out that a very large percentage of all areas of gingivitis are caused by operating which is directly abusive to the soft tissues, or which, on account of lack of care in the finer details, is indirectly injurious by permitting food lodgments and irritations. These have become so common that they are not noticed, or are given no consideration. It has been my constant endeavor in the writing of this book to impress the fact that, of the ordinary dental ills, nothing may lead to more serious consequences than one little area of gingivitis. The highest type of preventive treatment, therefore, will be the exercise of the finest care to do each operation so well that the soft tissues will suffer no injury. This may only be done as the result of a careful study and training by each man for himself. Every area of gingivitis presenting should be critically examined to determine its cause. The cause should then be removed and the case observed subsequently to know the result.

Injuries to the septal tissues. It would hardly seem necessary, after what I have said of the injuries to the septal tissues and the results, to call attention to them again. My main purpose is to refer to them under the discussion of preventive treatment.

The correction of the conditions which bring about the impaction of food in the septal spaces must, in its very nature, be done by the person who is also filling teeth. The regular practitioner of dentistry must be trained in the observation necessary to the early detection of these injuries and must be ready to correct the conditions giving rise to them at once. These conditions have been described. Fortunately there is usually sufficient time between the beginning of the impaction of food into a given space, and the time of serious injury to the peridental membrane, to allow for its discovery and treatment before the peridental membrane is permanently injured. The greatest element needing development is the ability to see these things and to act with reasonable promptness. If a worn contact in a given case is causing the lodgment of food in the septal space, one should be able to detect the shortening of the septal gingiva in good time to cut a cavity in one of the teeth, and build a filling with a prominent contact that will prevent food from passing, and to key up that part of the arch to the normal pressure of tooth against tooth. Then, as rapidly as possible,

the patient should bring heavy work in mastication on these teeth to increase the tonicity of their membranes. Usually no other treatment is necessary.

In other cases the treatment must be varied to meet the cause of the difficulty. In some cases, the cause of the inflammation will be obscure, and patient study will be required to determine it. But the rule is that the progress of injury will allow reasonable time for the discovery of the cause and its correction. Under no circumstances should such an injury fail to receive reasonably prompt treatment. Such a case begins to try the strength of the arch early sometimes, and if treatment is delayed, a number of other contacts may be leaking food, thus injuring the tissues in other septal spaces.

Injuries to lingual of upper incisors. Injuries to the lingual of the upper incisors, which have been considered, do not occur very frequently, but when they do, the causes usually become very distressing on account of the gradually increasing protrusion of the teeth, and *hopeless* in that they may not be bettered by treatment. For this reason very especial watch over these gingivæ should be kept up continuously. A watch for serumal calculus on the teeth within the subgingival space by frequent instrumental examinations should always be a habit of practice, because this calculus is covered by the gingivæ and is hidden from view. It does harm of a more serious nature in this particular place than elsewhere, but is liable to do serious harm anywhere that it may be lodged. It is an enemy in ambush that one should look for as a habit of practice. When found anywhere in a patient's mouth, it should be removed completely, and frequent examinations made at fairly regular intervals for its return. When it occurs once, it is liable to recur.

Training of patients. Patients should be taught to brush the gingivæ and cleanse the subgingival spaces as a part of the routine care of their mouths. In addition to the brushing, one of the best forms of treatment for keeping the gingivæ in good health is to wash them with a jet of water from the syringe. In those cases in which the gingivæ show too much redness, cases in which deposits of serumal calculus are most liable to occur, this treatment is very effective. I have found the tendency to continuous redness to abate more readily under the washing with the syringe than any other treatment which I have tried. In this cleaning, the tissues are in no danger whatever of injury. It seems to be especially suited to keeping the gingivæ throughout the mouth in a fine state of health, and it is worth

while to train patients in its use. The manner of doing this will be given in detail under Mouth Hygiene.

Palliative Treatment.

Palliative treatment should be applied to those cases in which detachment has occurred, but in which conditions are such that the effort is to be made to keep the cases under control. This generally means that such cases will require the best of care and watchfulness by both dentist and patient so long as the affected teeth remain in the mouth.

While it has been suggested that preventive treatment ends and palliative treatment begins with a suppurative detachment of the peridental membrane, yet the line between the two may not be drawn quite so sharply. As has been stated, there are many slight suppurations, which destroy a little of the attachment of the membrane, but which recover, except that there is no reattachment of the detached fibers. The gingivæ recede a trifle, the tissues accept a new line of attachment and there may be no further progress of the case.

Other cases occur in which a slight pocket remains as the result of a suppuration. The overlying tissue may not hug the tooth as closely as formerly, depending upon the extent to which the free gingivæ fibers have been cut off, and the space receives accumulations from the saliva, including pyogenic organisms. In addition, there is apt to be more than the normal amount of serum excreted, on account of the changed character of the tissue, and also on account of the irritation of the accumulations within the pocket. This is likely to bring a deposit of serumal calculus, which will increase the inflammation.

Pockets on labial and buccal surfaces offer the best opportunity for palliative treatment, because the teeth are less liable to move lingually, owing to the resistance of the curve of the arch; proper contacts may be maintained and the irritation caused by food impactions avoided.

Many cases may be held in abeyance for a long time by palliative treatment, particularly if only one or two teeth are involved. Sometimes the tissues in a certain interproximal space may be rather seriously injured, but the conditions are such that a good contact may be restored and maintained. If the pocket is not deep, or if there has been sufficient shrinkage of the septal tissue to reduce the depth, the case may be kept under control by the treatment which will be presented.

Plan for palliative treatment.

The plan for palliative treatment should consist: (1) The removal of deposits and care of the tissues by the dentist; (2) care by the patient; (3) subsequent examinations at stated intervals.

The effort should be to prevent pockets from becoming deeper, by clearing up the present suppuration and preventing a recurrence. Continued cleanliness of the pocket will more nearly achieve this end than any other plan of treatment. The dentist should remove the deposits, clean the pocket and otherwise put it in the best possible condition before turning the case over to the patient. Then the care by the patient should be that which will remove the accumulations, prevent new deposits as far as possible, and keep the soft tissue in the most healthy condition.

All that has been said in the treatment of inflammations caused by deposits of salivary calculus relative to the earnestness with which the dentist should arrange for the management of cases, the care in making and recording examinations, the impression which should be made upon the patient as to the importance of active coöperation, applies with equal force here. It should be borne in mind, and the patient should be brought to realize, that treatment is undertaken to prevent cases from progressing by preventing reinfections and further detachments, which means that each such pocket must be under practically continuous observation and treatment as long as the tooth remains in the mouth. It is a life-long task.

Removal of deposits and care by the dentist.

The removal of deposits of serumal calculus, which have occurred either upon the enamel of the subgingival spaces or upon the cementum within the pocket, is one of the important matters in the treatment of suppurative pericementitis. As has been said, a considerable number of cases present in which no deposits have occurred over long periods of time. However, the rule is that deposits do occur, although there is the widest possible variation as to the rapidity of their accumulation, depending upon the amounts of calco-globulin in the secretions, and also upon the quantity of serum poured out into the pockets. While deposits on the cementum necessarily occur after detachment, and are therefore not a primary cause of detachment, their presence serves to promote the progress of the disease, by increasing the inflammation of the overlying tissue. Therefore,

the first indication in treatment is for the removal of these deposits.

For cases in which a number of teeth are involved, the thorough removal of the deposits requires the most painstaking technic and the limit of persistence. Attention should be given to a particular section of the mouth, or to a few teeth, at a sitting, in order that no particle of deposit may be missed. This should be done in a systematic way, to include every denuded surface.

Instruments. The technic of removal is practically the same as that already mentioned for the removal of deposits from subgingival spaces, except that as pockets are deeper, the difficulties in manipulation are increased. The instruments to be used are the same. The set of scalers shown in Figure 266 was designed primarily for the purpose of reaching the various surfaces of roots in doing this operating.

The accompanying radiographs show several of these instruments in their proper positions. (See Figures 268 to 284.) With all of the instruments it will be noticed that the working points — the blades — are practically in line with the handle, which is an essential feature to accurate manipulation. (See Figure 265.) The greatest length of blade is 8 millimeters (nearly one-third of an inch); this is considered about the limit of depth of pocket in which this plan of treatment should be employed. Certainly in most cases pockets of this or even less depth will be kept under control with much difficulty, and many teeth with pockets of this depth should be extracted.

There are in this set twelve instruments. Two of these are peridental membrane explorers. They are especially designed for feeling the line of attachment of the peridental membrane to the cementum. These have smoothly rounded ends, so that they will not injure the tissue, and may be carried around each root with the end following the line of attachment of the tissue. (See Figure 268.) The rougher deposits may be easily detected with these instruments, although sharp blades should be used to determine the exact condition as to finer deposits. There is a special pair of explorers, not included in the regular set, made with longer blades, also with smoothly rounded ends; these are graduated with a scale of fifteen millimeters, beginning at the end. (See Figure 267.) In making examination charts these instruments are used to record the depth of pockets, as will be explained under the heading of Examinations of the Mouth.

There are six pull scalers, in three pairs, each having blades 1.5 mm. wide and 8 mm. long, but of different angles. These are all made with curved blades, somewhat similar to spoons, but with square ends. The blades of one pair are straight, as viewed from one direction, but have a very slight curve as viewed from the other direction. One has the cutting edge *looking toward* the handle, the other *looking away from* the handle. The second pair has the formula 15-8-6, and the third pair 15-8-12; these having blades at angles of 6 and 12 centigrades, respectively, to the handles. There is one pair of push scalers with the formula 15-8-12. In addition there is a sickle form and a cleoid form, which are designed to reach shallow depths on the proximal surfaces of front teeth.

Instrumentation. In the upper incisor region, the straight instruments may be used for the major part of the operating on all four surfaces of each root, although those of 6 centigrade angle will occasionally be found more convenient for proximal surfaces. For the upper bicuspids the same instruments may generally be used, although those of 6 centigrade angle will more often displace the straight blades. (See Figures 269, 270, 273 and 274.)

In the upper molar region the 12 centigrade blades will generally be most convenient. In many mouths the 6 centigrade or the straight blades may be used on both buccal and lingual, and in a fair percentage on the distal surface of the first molar. Sometimes the second molar may be reached with these instruments. The straight pull instrument with the blade looking toward the handle is to be used on distal surfaces. The corresponding instrument, with the blade looking away from the handle, is especially well adapted for reaching the region of mesio-lingual angles of the roots of these teeth. (See Figures 275 to 278.)

For the lower incisors, as for the upper, the straight instruments may be used, although those of 6 centigrade angle will be more often found desirable, particularly at the mesio- and disto-lingual angles. The sickle blade or the cleoid may often be used in this position. (See Figures 271 and 272.)

For the lower bicuspids and molars, the 12 angle blades may frequently be used on all surfaces, although the 6 angle will often be more convenient for the bicuspids. On all of the lower bicuspids and molars, the straight instrument with the blade looking away from the handle will be found to reach the

mesio-lingual angle better than any other. (See Figures 279 to 284.)

Finger skill very essential. This set of scalers is considered sufficient in number and variety of form to reach all positions in pockets which should receive palliative treatment. In fact a less number will be sufficient for the more skilful operators. The most essential thing for the thorough scaling of roots is the development of proper finger skill. Dentists who have not trained themselves so that they are able by the sense of touch to find each particle of deposit, and to so locate it that the instrument may be properly placed for its removal, will not be successful in scaling operations, even though they have the largest conceivable number of instruments. This is a skill acquired by long training, much the same as the training required to master the finer technique of the piano.

One will do well to occasionally test his ability in this operating, by performing the most thorough possible scaling of a root which is to be extracted, going over the denuded surface again and again, until every particle of deposit is apparently removed. An examination of the root after the tooth is extracted will reveal the thoroughness or lack of thoroughness of the technic employed, and will indicate the measure of success which is being attained in similar operations on teeth which are retained.

Scalers must be sharp. It is absolutely essential that scalers be sharp if the deposits are to be successfully removed. The deposits are usually very closely adherent to the cementum and it requires both a very sharp blade and considerable force, accurately applied, to remove them. Isolated nodules may generally be removed with less difficulty than the flatter scale-like deposits. In cases of long standing almost the entire cementum of the pocket may be covered, and the deposit may be in the form of a tolerably smooth mass, or more or less roughened. The removal of such a deposit is much more difficult than of the separate nodules. This requires the most careful technic and persistence and the employment of the sharpest possible blades. These instruments must be delicate and of such form that they may be manipulated within the pocket, without unnecessary injury to the soft tissues. The blades should be sharpened often, but should not require more than a movement or two on the oil-stone with light pressure. The blades of the pull scalers, particularly, may be worn away in a short time by too much

grinding on the stone. The pull scalers must be frequently replaced by new instruments.

As a rule, the pull scalers will cause less injury to the soft tissues than the push scalers, especially by other than the most skilful operators. The pull scalers may be very carefully carried to the depth of the pocket, the edge of the blade being held against the surface of the root, feeling the deposit as the blade passes over it, until the movement is stopped by the end of the instrument coming in contact with the attachment of the membrane. Then the blade should be held hard against the root and drawn out in an effort to bring the deposit with it. These instruments are used as hoes. If the instrument does not bring the deposit away, the failure is usually due either to the fact that the blade is not sharp, or that it was not held in proper relation to the root, or the force applied was insufficient.

In the use of the push scalers, the edge of the blade is placed on the enamel near the gingival line, and the instrument, while held close against the root, is carried toward or to the full depth of the pocket, the attempt being made to cut the deposit away from the root with the movement. The finger position should be such that the pushing movement is under complete control by a rest of fingers on neighboring teeth, or possibly by a finger of the opposite hand, to prevent the instrument from plunging into the soft tissue as the deposit breaks away, or if the instrument should slip. In positions in which there is good access and opportunity for perfect control, these instruments are more effective than the pull scalers. The angle of bevel of the blades is such that they will hold their edge better, and remain sharp longer.

Leave roots smooth. The effort should be to remove all of the deposit and leave the surface of the root as smooth as possible. Some writers have contended that a portion of the cementum should be planed off in the effort to remove all of it that had become saturated with the products of the suppurative process. There are several reasons why this seems to me to be impractical. The cementum is so porous that, in cases in which the infection has been in progress for a time, it will become saturated with the products of suppuration. Even in very recent cases, if it were possible to know when one had removed the infected portion, we could not expect to get a normal reattachment, because of the disappearance of the cementoblasts and fibers from the detached tissue. The case would soon become reinfected and the previous condition re-established. Even if

in rare instances an attachment, such as is obtained in cases of planted teeth, should occur, this has been shown to be physiologically unstable. The excessive planing of the cementum will often render vital teeth hypersensitive, making future scaling operations very painful. Such hypersensitiveness of a single tooth may reflexly cause the formation of secondary dentin within all of the teeth.

In many cases the cementum of a pocket is considerably softened by the growth of acid forming micro-organisms within the pocket. The acid dissolves the calcium salts from the cementum, softening it in the same manner in which dentin is softened by caries. Cementum in this condition is very easily removed with a sharp instrument.

Pain in scaling operations. Pain in connection with scaling operations is caused in some cases by the sensitiveness of the pulp of the tooth and in some on account of the inflammation of the peridental membrane. There is no physiological provision for the transmission of sensation through the cementum. The ends of the dentinal fibrils, just beneath the cementum, must receive some stimulation for sensation to be conducted through to the pulp. Thermal changes may be sufficient to cause pain in cases where the recession of the gum has exposed the cementum, or in the irrigation of pockets with water which is too hot or too cold. Usually pain caused by instruments in scaling is the result of the removal of the cementum, exposing the surface of the dentin. The area of dentin immediately below the cementum is more sensitive than closer in toward the pulp, because of the branching of the ends of the tubules, presenting many fine sensitive filaments in this portion of the dentin.

In cases in which there is continual complaint of pain on account of the sensitiveness of teeth with pockets, the only remedy for such pain is to remove the pulp. This, of course, relieves the difficulty at once. However, pulps should not be removed without a definite indication for so doing. The practice of removing pulps, which has been recommended as being beneficial in the treatment of suppurative pericementitis, is unwarranted.

Pain within the peridental tissues in connection with scaling operations is usually due to hypersensitiveness or to abuse of the tissues. The hypersensitiveness may be reduced by thorough irrigation of the pocket on two or three successive days, or a solution of novocain may be injected into the surrounding

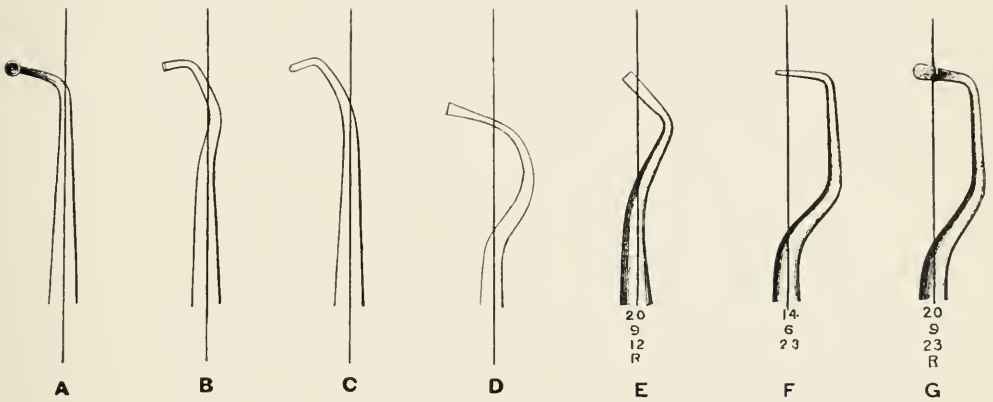

Fig. 265.

Fig. 265. A, B, C and D, Instruments wrongly contra-angled. Their points are so far from the line of the central axis of the shaft that they incline to twist, or turn, in the fingers when the effort is made to cut with them. They are out of balance. E, F and G, Instruments correctly contra-angled. Points are brought close enough to the line of the central axis so that they will not be inclined to twist, or turn, in the fingers when the effort is made to cut with them. They are well balanced.

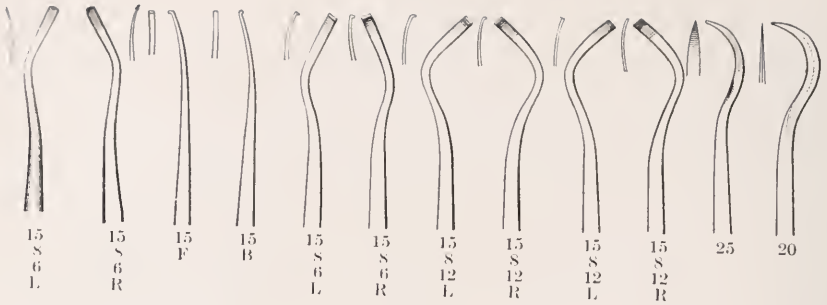

FIG. 266.

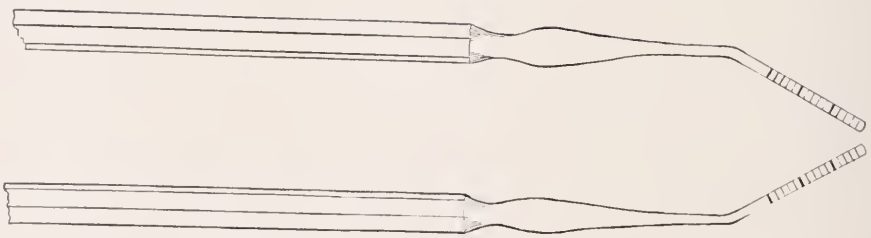

FIG. 267.

FIG. 266. Set of scalers for removing deposits of serumal calculus. These are described in the text, and some of the positions in use are shown in Figures 268 to 284.

FIG. 267. A pair of special explorers for measuring the depth of pockets. A millimeter scale is cut in the blades. It is not necessary to see the blade while in the pocket to make a sufficiently accurate reading; the serum within the pocket will adhere to the blade and the reading may be made after the instrument is removed.

FIG. 268.

FIG. 269.

FIG. 270.

FIG. 271.

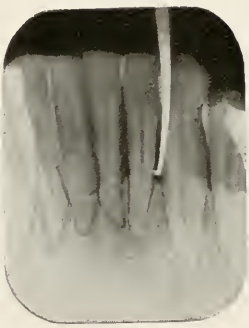

FIG. 272.

FIGS. 268 TO 284. A series of radiographs illustrating the use of the scalers in removing deposits of serumal calculus. The pockets in this case were too deep to require scaling operations, as extraction of all of the teeth was indicated. This case was used to better illustrate the instrument positions.

FIG. 268. This shows one of the peridental membrane explorers which has been carried down to the attachment of the soft tissue to the cementum on the labial side of a lower lateral incisor. The rounded end of the instrument is nicely shown.

FIG. 269. The almost straight pull scaler, with blade looking toward handle, in position on the distal surface of an upper lateral incisor.

FIG. 270. The almost straight pull scaler, with blade looking toward handle, in position on the mesial surface of an upper central incisor.

FIG. 271. The 15-8-6 pull scaler in position at the mesio-lingual angle of a lower lateral incisor.

FIG. 272. The almost straight pull scaler, with blade looking toward handle, in position on the distal surface of a lower lateral incisor.

FIG. 273.

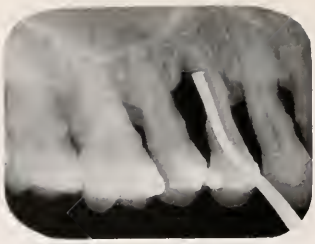

FIG. 274.

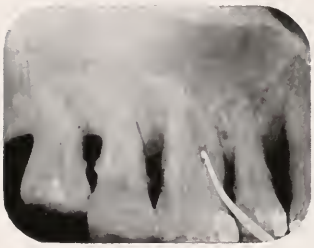

FIG. 275.

FIG. 276.

FIG. 277.

FIG. 278.

FIG. 268 TO 284. A series of radiographs illustrating the use of the scalers in removing deposits of serumal calculus. The pockets in this case were too deep to require scaling operations, as extraction of all of the teeth was indicated. This case was used to better illustrate the instrument positions.

FIG. 273. The almost straight pull scaler, with blade looking away from handle, in position at mesio-lingual angle of upper second bicuspid.

FIG. 274. The 15-8-6 pull scaler in position on lingual surface of upper first bicuspid.

FIG. 275. The almost straight pull scaler, with blade looking away from handle, in position on mesial surface of lingual root of upper first molar.

FIG. 276. The almost straight pull scaler, with blade looking toward handle, in position on distal surface of upper first molar.

FIG. 277. The 15-8-12 scaler in position on lingual surface of upper second molar.

FIG. 278. The 15-8-12 scaler in position on buccal surface of upper first molar.

FIG. 279.

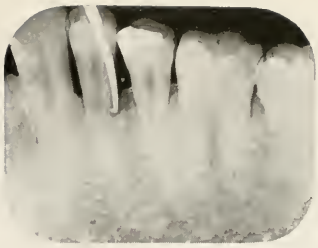

FIG. 280.

FIG. 281.

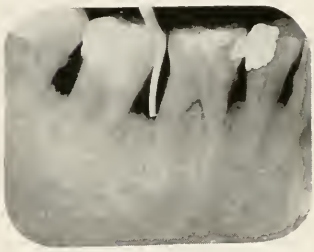

FIG 282.

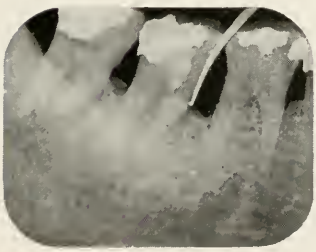

FIG. 283.

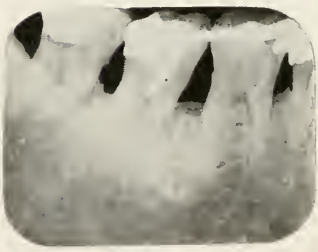

FIG. 284.

FIGS. 268 TO 284. A series of radiographs illustrating the use of the scalers in removing deposits of serumal calculus. The pockets in this case were too deep to require scaling operations, as extraction of all of the teeth was indicated. This case was used to better illustrate the instrument positions.

FIG. 279. The 15-8-6 pull scaler in position on the mesial surface of the lower first bicuspid.

FIG. 280. The almost straight pull scaler, with blade looking toward handle, in position on distal surface of lower first bicuspid.

FIG. 281. The almost straight pull scaler, with blade looking toward handle, in position on distal surface of lower first molar.

FIG. 282. The 15-8-12 scaler in position on distal surface of lower first molar, approach being through the buccal embrasure.

FIG. 283. The almost straight pull scaler, with blade looking away from handle, in position on mesio lingual of lower second molar.

FIG. 284. The 15-8-12 scaler in position on buccal of lower second molar.

Fig. 285.

Fig. 286.

Fig. 285. Plaster model of case in which the tissue overlying a pocket on the mesio-buccal root of an upper first molar was cut away to reduce the depth of the pocket and facilitate the cleaning.

Fig. 286. Plaster model of a similar case on the buccal side of the mesial root of a lower first molar.

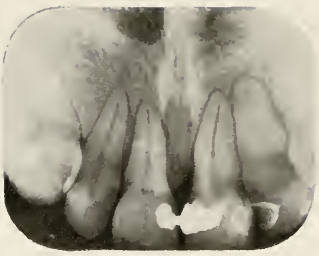

FIG. 287.

FIG 288.

FIG. 289.

FIG. 287. Radiograph of a case in which an injury with a ligature had caused the formation of a pocket on the mesial side of the upper left central incisor and the teeth had separated about 1 mm. They were gradually drawn together, and the appliance shown in the illustration was cemented in place (on the lingual) to hold them in contact. This was in October, 1909. The radiograph was taken five years later.

FIG. 288. Radiograph of a case in which the alveolus of a lower second molar has been almost entirely destroyed.

FIG. 289. A panoramic radiographic view of a case of suppurative pericementitis. This patient, a woman of forty, had complained for several years of joint inflammation, particularly of the elbows, knees and ankles. The lower molars had all been extracted. The lower incisors and cuspids were in good condition, the bicuspids were much involved.

FIG. 290.

FIG. 291.

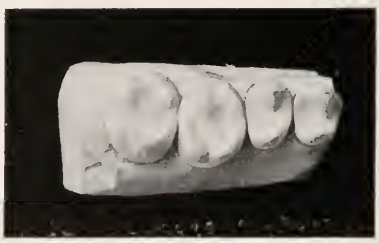

FIG 292.

FIG. 293.

FIGS. 290 TO 293. Plaster models of cases in which roots were amputated.

FIG. 290. The lingual root of this upper first molar was amputated on account of a suppurative pericementitis. The tooth was slightly elongated at the time. The impression for this model was taken fifteen years after the operation. The soft tissue hugs the remaining roots very closely and the contour is such that it is easy to keep clean.

FIGS. 291 AND 292 are buccal and occlusal views of the same case. The mesiobuccal root of this upper first molar was amputated and a gold crown placed on the other two. The soft tissue fills the septal space very perfectly.

FIG. 293. A case in which the distal root of a lower first molar was amputated. The distal half of the crown of the tooth was also cut away, and a gold crown was made to restore the full occlusal surface. The patient cleanses the open space by flushing it with a mouthful of water.

tissues and the operation may proceed at once without discomfort to the patient.

Care of tissues by the dentist. What has been said relative to the care of the gingivæ following the removal of deposits of serumal calculus applies with even greater force to the tissue overlying pus pockets. In connection with the removal of the deposits the pockets should be thoroughly irrigated with warm salt solution, to remove small particles of deposits which may not have been brought away with the scalers. This will also cleanse the space of blood and other debris, and leave the tissue in the best possible condition. This will be referred to in the discussion of the reasons for the abandonment of the use of antiseptics in these pockets.

The patient should be required to return several times, if necessary, at intervals of a few days for irrigation of the pockets. This should be continued until the inflammation is reduced and the mouth and teeth are comfortable. The patient's attention should be called to the technic of using the syringe and he should come to feel the washing of the pockets in order that he may know that he is succeeding in the subsequent care of his mouth.

Care by the patient.

One could hardly conceive of better care on the part of the patient than the washing of such pockets twice daily, using salt solution in a rubber bulb syringe. This is effective in that the space is thoroughly cleaned without injury to the soft tissue. The future of such a case depends very largely on the care which the patient may be induced to exercise. Those who are faithful may so fully control conditions that little progress will be made in many years. For all practical considerations, such cases are well so long as they are kept clean. Careful and persistent washing will keep the tissues free from irritation caused by accumulations, and all micro-organisms which are free in the pocket will be removed at each washing. Under this treatment the progress of such cases may be stayed, and the teeth may be retained for many years of comfortable service, without danger to the general health. Proper care by the patient can not be expected unless the need of it is explained and the patient's interest is awakened. Exact directions should be given, and it should be urged that these be carried out to the most minute detail.

The syringe shown in Figure 187 is the best type for the

patient. The bulb fits well in the hand. If the patient is instructed to hold the nozzle between the thumb and first finger while the bulb is pressed with the other fingers, he will have better control of the position of the end of the nozzle. They must learn to hold it in just the right relation to the teeth and the gingivæ, as shown in Figure 188.

The sodium chloride tablets, which can be purchased at almost every drug store, are very convenient for this purpose. An ordinary drinking-glass holds about eight ounces of water, which requires two tablets to make a physiological salt solution. (See Figure 177.)

In some cases in which proximal surfaces are involved and the septal tissue has receded, contacts can not be maintained tight enough to prevent a little food from passing occasionally, although this may not become crowded very tightly upon the septal tissue. The septal space will, however, fill up by food entering it from the buccal and lingual embrasures, rather than through the contact. In this case the food is not packed very tightly and does no great harm if it is regularly removed after meals.

Generally such impactions can be cleaned away by the syringe alone. Sometimes a toothpick may be necessary to remove them. It is to this class of cases particularly that the wood toothpick is most applicable, for in these there is plenty of space in which to use the broad end of the toothpick without injuring the remaining tissue. There are a good many cases occurring in elderly persons in which the septal gingivæ are practically gone, and the spaces between the gingival portions of the proximal surfaces fill with food from the embrasures, when there is no leak between the contact points. This is best removed by the wood toothpick followed by the jets of water from the syringe. I have seen cases of this kind go on doing good service for many years with this simple treatment.

Subsequent examinations.

Patients should be required to visit the dentist frequently for examination, and for the correction of any errors that may be found in their own care. As previously mentioned for other conditions a definite arrangement should be made for the care of the case. It should be understood that this is necessary to success. By such a plan, beginning pus pockets may be found at a very early period, so that palliative treatment can be continued against these to the best advantage. The key to all of this

palliative treatment is the holding in check of suppurative processes. If this be successfully done, the palliative treatment is worth while; if in any case it is not successfully done, the palliative treatment should be discontinued, and the teeth should be removed.

The administration of emetin hydrochlorate.

In discussing the causes of suppurative pericementitis I have referred to the discoveries of Barrett and others of the endameba buccalis in practically all of these pockets. While these findings have not yet proven that these protozoa are the cause of the formation of the pockets, there seems to be no question but that the use of an endamebacide results in a prompt improvement in the appearance of the overlying tissues. Emetin hydrochlorate, an alkaloid of ipecac, is administered for this purpose, either by injection into the pockets, by tablets taken by the mouth, or by hypodermic injection in the arm or other convenient location — one-third or one-half grain may be given each day for four or five days.

While it now appears likely that this very simple treatment will be a valuable aid in the management of these cases, its use must in all probability be considered as a palliative rather than a curative measure, and its effect very temporary, as the pocket will remain as a harbor inviting reinfection. As has been mentioned, the investigations of the role which the endamebas play in this disease have not yet progressed sufficiently to justify an extended report in this book. This discussion is better suited to journal articles for some time. The work which has been done is, however, one step further in our study of the pathology of this disease.

Surgical treatment of pockets.

In cases in which the pockets on labial, buccal or lingual surfaces are deep, the depth may be reduced by cutting away the overlying tissue, or, in other words, by cutting away the gum tissue that is undermined by the disease of the peridental membrane. This should be cut away as far, or a little beyond, the point to which the disease has reached, which generally means some cutting away of the margin of the alveolar process as well as the soft tissue. In this way the pocket can be eradicated, and in some cases a fairly permanent cure can be effected, though this is rare. (See Figures 285 and 286.) A very good way of doing this is with the actual cautery. If the wire is white

hot, it cuts very nicely, without causing much pain, and with very little hemorrhage. Many of the cauteries now being supplied to dentists do not have sufficient current to heat the wire as hot as it should be; therefore they cause unnecessary pain and do not cut well.

The tissue may be cut away with a knife, novocain being injected for anesthesia. A tenaculum may be thrust through the tissue into the pocket, and while holding with this, two cuts should be made parallel with the length of the root and one connecting these at the position of the deepest part of the pocket; or the tissue forming the pocket may be held with a pair of pliers or tissue forceps, one beak being placed within the pocket, while the cuts are made with the knife. The tissue must be very thoroughly removed, and even then it will often grow over a portion of the denuded root, reforming the pocket. The wound should be thoroughly irrigated until all hemorrhage has ceased, and no blood clot should be allowed to remain, as it will aid the tissues in building across the space. It will often be necessary to cut this away several times.

There is considerable variation in results following this treatment. In some cases there will not be much tendency to the reforming of the pocket, or there will remain a very shallow pocket in place of the former deep one. The shallow pocket gives the patient the opportunity to do more effective cleaning and the danger of reinfection is therefore very greatly reduced. In other cases the tissue will regrow, covering all or nearly all of the space from which it was cut, and little or nothing will have been accomplished. The rule is, however, that something will be gained in reducing the depth of the pocket each time the overlying tissue is cut away.

This treatment is often contraindicated in the upper incisor region on account of the unsightly appearance of the denuded root. However, if it becomes a question of exposing the root, or extracting the tooth, the loss of the tooth may be postponed by cutting off the overlying tissue.

There is little hope for those cases in which pockets of considerable depth have formed on proximal surfaces. It is usually impractical to cut away the interproximal soft tissue to eliminate a pus pocket, as has just been mentioned for buccal and labial surfaces, as there would remain a pocket between the teeth which would be little better than the previous pus pocket. Occasionally cases present in which several teeth in the bicuspid and molar region have had their membranes detached for about

an equal distance on proximal, buccal and lingual surfaces. If the pockets are not very deep, the soft tissue may be removed all around such teeth, thus leaving the interproximal spaces open to the gingival of the contacts, the remaining interproximal tissue being on the same level as that on the buccal and lingual. This gives the best opportunity for cleaning.

Following such an operation, the condition is similar to that occasionally seen in the mouths of old people, the gingivæ having receded sufficiently to leave all of the enamel and sometimes a little of the cementum exposed to view, with a considerable open space to the gingival of each contact.

The use of splints.

In some cases splints of various forms may be used to prevent tooth movement. If there is a single pocket which has progressed sufficiently to cause the contact to be opened, a splint may be applied to hold the two teeth firmly together and thus prevent food impaction through the contact, and also prevent the opening of other contacts. Two gold bands, soldered together, may be cemented on. Such an appliance is particularly well adapted to lower incisors. In the bicuspid and molar region, two gold inlays, soldered together at the position of the normal contact of the teeth, may be employed. Or, in the case of the upper incisors, an appliance may be placed on the lingual surfaces, if the bite will permit.

I show a radiograph (Figure 287) of a case in which a pocket about three millimeters deep on the mesial surface of an upper left central incisor, had caused the teeth to separate about one millimeter. This pocket had been caused by drawing a ligature too tightly about the tooth, cutting away the fibers on the mesial surface. Several days were required to bring the teeth into contact. Then two holes were drilled into the lingual surface of this tooth in the gingival third. A slot was made in the enamel connecting them, and an inlay was cast to fit the slot and holes. A single hole was cut in the right central, and a piece of iridio-platinum wire, bent to fit in this hole in the right central, was soldered to the inlay. The appliance was cemented to place, holding the teeth in contact. This operation was performed in October, 1909. The pocket has received careful attention since and the depth has not increased more than one and one-half or possibly two millimeters in the five years which elapsed before the radiograph was made. The patient, a woman of forty, has never had a tooth extracted and there is no other

disease of the investing tissues about her teeth. She is very faithful in the use of the syringe.

Such measures may be indicated in cases like the one just cited, but be contraindicated in others where the pockets are no deeper. The greater the number of pockets, the less should we think of stay appliances, and the more of extraction. The difficulties and dangers increase with the number and depth of the pockets. In those cases in which there is a more or less general involvement of the investing tissues, the value of the teeth for mastication must be weighed against the danger to the general health, and I am inclined to advise radical rather than palliative treatment.

Radical Treatment.

When indicated. Radical treatment by root amputation or extraction should be employed in all of those cases in which the teeth are not doing well under palliative treatment. In cases in which periodic severe suppurations occur, and especially those in which the patient has a slight rise of temperature much of the time, or if there is considerable soreness of the cervical lymphatics, palliative treatment should be discontinued. These conditions should not be allowed to continue because of the danger of systemic infections. It will be a surprise to many how quickly the cervical glands, which are sore when palpated, disappear or become very small nodules which are difficult to find, after the teeth have been extracted. The entrance of infection into the system through the lymphatics is stopped at once.

It is well to keep a close watch over the temperature of these patients. If the temperature is found to run from 99 to 100 degrees, it should be taken as an indication that a slight toxemia is occurring from the absorption of the products of bacteria which are growing in the pus, or from the pus which is being absorbed.

The discussion of systemic infections from mouth foci is presented elsewhere. What I wish to urge here in the management of cases is that we should be very certain that we do not allow general or special infections of distant parts which will menace the general health and life of the patient. The application of radical treatment will put a prompt stop to all of this, for the parts heal very readily indeed after operations of this character. If any single statement can be made with greater positiveness than all others, relative to this disease, it is that the suppurative condition is cured almost immediately with the

extraction of the tooth, so far as each particular socket is concerned.

As pockets present which are deeper, the difficulties increase. As soon as those fibers which pass from tooth to tooth, or from tooth to alveolar process are involved, tooth movement is apt to begin. This movement is more serious when the involvement is on the lingual of the upper incisors, or on the proximal surfaces, particularly of the bicuspids and molars. In either case the gradual movement and separation of the teeth, which have been described in detail, present the greatest difficulty in the management, because the opening of the contacts invites food impaction, which causes sufficient irritation to keep up a low-grade inflammation, even though the patient gives the best possible care by prompt and thorough cleansing.

Over and over again dentists come to me with models of protruding incisor teeth, and ask my advice as to moving them back and the possibilities of keeping them in place. Generally, I advise that they be extracted. If this is done in good time, it may save the remaining teeth. If only the four incisors are involved, and these are extracted, a bridge supported by the cuspid roots may be placed with fair prospects that it will do good service. I have followed this plan in a considerable number of cases in which the curve of the arch was not too great. I recall one case, in which I did not consider it wise to place a bridge, because the leverage would be too great on the teeth to which it would be attached, and I made a partial denture carrying the four teeth. This covered the entire roof of the mouth, and for many years afterward there was no involvement of the membranes about the remaining teeth.

Radical treatment, then, means that each tooth that has given rise to a considerable pus formation, which can not be controlled by palliative treatment, should be extracted as soon as this fact has been ascertained. It should make no difference whether there are two or three teeth together, or the whole number of teeth in the mouth. It is wrong from several points of view to allow such cases to go on suppurating year after year, not only on account of the menace that such a suppuration is to the life of the individual, which is enough in itself to demand the extraction of such teeth, but also from the fact that the continued suppuration until the teeth have loosened, cuts away so much of the alveolar process, and even of the bone in which they are seated, that when the teeth are finally lost there is practically no residual alveolar ridge on which to place a set of artificial teeth

that may be used with comfort. After watchfulness for many years, I have come to consider that this is really a very important matter, and always when suppuration has gone on until it is plain that no cure can be had in the case except by radical treatment, the teeth should be extracted without further delay. It is very bad practice indeed to treat a case until finally, when the teeth have been extracted and the mouth has healed, it is found to be impossible to make a set of artificial teeth which may be worn with comfort and be satisfactory in mastication, because of the very scant residual alveolar ridge. The patient is then doomed to trouble for the rest of his lifetime, and this could have been prevented if the teeth had been extracted in time.

I should not, however, continue very loose teeth in the mouth under any consideration. When teeth have become shaky on account of the extensive parting of the membrane from them, they should certainly be extracted without delay. The fact that some such teeth have been known to tighten and do service should not enter into practical consideration, for the cases have too much uncertainty to enter into a rule of practice. Figure 288 shows a lower second molar which was held to the process by but a few fibers of the peridental membrane about the apex of the root. Many other illustrations show teeth with very little attachment remaining. In Figure 261 there is hardly a tooth in the entire denture which has sufficient attachment to hold it securely in place.

Once, in visiting a dentist who was enthusiastic in the treatment of these diseases, I sat waiting for him to complete a treatment of a case. I knew the patient and knew the condition of his mouth. After the dentist was through, he came into the reception-room with this patient and I talked with them for a few minutes. When the patient had gone the dentist asked me a question regarding the case and I insisted that he should extract every tooth in the mouth without delay. He replied that he would cure the case and I needn't be alarmed. I was next reminded of this case by the announcement in the papers that the man had died of heart disease coming on after an attack of rheumatism. This man would not have been classed as a very susceptible person to disease, but rather the contrary. He had always been a robust, healthy person. My belief is that the mouth infection was the cause of his death.

I have seen similar cases among patients whom I have myself observed and examined, and some who have been under my care. I tried the treatment which I believed to be best, as

thoroughly as possible, until some of these cases were lost by death occurring somewhat suddenly, as in the case mentioned. Others, I think I saved by the liberal use of the forceps, and they became healthy and vigorous again.

Amputation of roots.

The lingual root of the upper first molar is often hopelessly involved in disease, when no other root in the mouth has lost its membrane. The rule is that this root may be cut away from its crown, and the tooth left standing upon the buccal roots. If these are healthy, it may do service for many years. My own experience in this operation has been especially fortunate, for I can recall no case in which such a tooth has not done well, if the disease was confined to that one root. The amputation is done by cutting the root away close against the crown of the tooth, aiming to cut it at the bifurcation of the roots, with a good slope toward the occlusal, which will clean readily. The space laid open in this way will often so fill up with soft tissue that no pocket will remain. Of course, before this is cut away, the pulp must be removed and the root canal in this particular root should be solidly filled, preferably with gold. This may be done in a few minutes and leaves a filling in the canal after the root is amputated, which may be nicely polished. Figure 290 is from a model made fifteen years after the lingual root of an upper first molar was amputated.

This operation may be applied to any one root of the upper first molar. If the mesio-buccal root has a pocket on its mesial surface which has become so deep as to be incurable, and in which palliative treatment is not successful, it may generally be cut away after a similar fashion and removed. This will end the danger of infection at once. Figures 291 and 292 are of an upper first molar, the mesio-buccal root of which was amputated. A gold crown was placed, restoring the full occlusal surface. I have cut away the disto-buccal root a number of times, yet it is not quite as easy of approach as either of the other two. It is often much depressed in between the other roots at its junction with the crown, and this makes the operation somewhat more complicated. A close study of the case beforehand will show the direction of the cutting necessary, and it may be accomplished with safe-ended fissure burs, insuring that there will be no cutting of other roots by contact of the end of the instrument. I have in a few cases cut away both buccal roots. Occasionally a similar operation may be done on

an upper second molar, although the bifurcation of the roots is seldom low enough, and the roots usually stand too close together to make it successful. In most cases, a radiograph will show the position of the bifurcation and the relation of the roots. Or curved explorers, such as are used in examinations for proximal decays, may be passed alongside the root and around it at the position of the bifurcation.

Either the mesial or distal root of a lower first molar may be similarly cut away, and the tooth will do well with a single root. Sometimes a root of a lower second molar may be removed in the same way. In amputating one root of a lower molar, I have usually cut away the corresponding portion of the crown. The cutting to separate the roots should be entirely from the root to be removed, thus leaving the remaining root in the best form to receive a crown. Figure 293 is from a model of a case in which the distal root of a lower first molar was removed. The entire occlusal surface was restored with a gold crown, which had been in place nearly seven years when the impression was taken.

Management of Cases of Chronic Suppurative Pericementitis.

Examination.

The determination of a course of treatment for each case presenting should be based upon a careful examination of the teeth, gingivæ, gums and alveolar processes. A visual examination should first be made, noting any points of redness or other signs of inflammatory movement; also looking for the extrusion of pus in any position, and especially pus exuding from between the gingivæ and the teeth. Deposits of salivary calculus should be noted, also any other causes of inflammations.

When satisfied with the visual examination, then a more close and careful examination should be made by palpation and instrumentation. A finger should be placed on the gum on either side of the alveolar process and with considerable pressure gradually brought down onto the gingivæ, over each tooth, beginning at some certain point such as a central incisor, and following back tooth by tooth to the third molar, watching for the extrusion of pus from beneath the gingivæ while so doing. Pus issuing from between the gingivæ and the tooth is a special mark which should be carefully noted, whether it comes from the lingual, from the buccal, or from the proximal side. This examination should include every tooth in a regular order, often

repeating the movement about a certain tooth to be sure of correctness. In doing this the sense of touch should be used very carefully to detect any points from which the alveolar process may have been absorbed about a particular tooth. This part of the examination is especially important, and every dentist should carry himself through a very severe training in order to become proficient. Each tooth should be tested as to its firmness or looseness and inquiry should be made as to the cause of the loss of missing teeth.

An instrumental examination should next be made. The subgingival explorer should be used; beginning in some certain place, the attachment of the gingivæ to the tooth should be explored in its entire circumference, so that it may be definitely known whether the attachments are all perfect, or if there be breaks and pockets along the sides of the roots. If there are pockets, the depth of the pockets becomes very important. A careful search should be made for deposits of serumal calculus, both in subgingival spaces and pus pockets. This may be done with the subgingival explorers at the same time when the line of attachment of the peridental membrane is being determined, although in extensive cases a separate examination should be made for each to insure greater accuracy. In this way, tooth by tooth, the whole mouth is examined.

An examination should be made to determine, if possible, the cause of each area of inflammation. In many instances this will include the condition of the contacts and will require that they be carefully tested. The relationship between the contacts and depth of proximal pockets is of importance in determining whether or not operations for the restoration and maintenance of proper contacts would be successful.

A record of the examination should be made which should include every point that is not in health, and the cause, or causes, if ascertained. As skill is acquired in making these examinations, they can be done quite rapidly, and yet thoroughly. A simple plan of doing this will be given under the heading of Examinations of the Mouth.

Radiographs. The examination should usually include a number of radiographs — the number depending on the preceding findings. If but a single pocket is found, and all the other tissues are in good health, a radiograph may not be considered necessary, particularly if a local cause of the pocket is evident. As the number of areas of inflammation is larger, the necessity

for radiographs is increased, both for the purpose of confirming the instrumental examination, and to show definitely the extent of absorption of bone which may have occurred. In all cases in which a considerable number of teeth are involved, the radiographs should include the roots of every tooth in both arches. One will often be surprised in such cases to find a much more extensive involvement of the alveolar process than is indicated by careful examinations without radiographs.

Radiographs are necessary to a full history of a case. They should be taken on the small films placed inside the mouth. This usually requires ten for two full dentures; two on each side and one in front for each arch. At the present time, the transparent (positive) films seem to give the best results. They are read best by holding a ground glass between the film and a light, although they show nearly as well when held toward a window. Thicker films, which require the making of a print, are less desirable, as the finer detail is not so accurate. Plates, which include a larger number of teeth, generally show insufficient detail to be satisfactory.

If a case of suppurative pericementitis is found and recorded by radiographs in this way, the progress of the disease may be readily noted by taking additional radiographs in the future, and comparing them. These radiographs should be squarely through the interproximal space, so that a pocket on the proximal surface will be clearly shown. If not squarely through the interproximal space, the tooth itself may obscure the pocket. Sometimes it will be found necessary to make two radiographs of the front teeth, one for each side of the median line, in order to get the direction right to show pockets on the mesial or distal sides of the roots of these teeth.

The best radiographs will show the destruction of bone to the labial, buccal or lingual of roots, although in many cases the density of the root obscures the picture. In cases in which there is some doubt as to whether the radiograph shows the true condition, a subgingival explorer may be passed to the full depth of the pocket and held in position while the radiograph is made. This instrument will be shown through the tooth and will indicate in the radiograph very accurately the depth of the pocket.

Plan of treatment for each case.

The treatment to be followed in the particular case may be either preventive, palliative or radical, or a combination of these methods.

Cases will vary from those in which only a few teeth are slightly involved, requiring palliative treatment, to those in which the denture is already so badly wrecked that immediate removal of all teeth will be determined to be the best course. In many, certain teeth with bad pockets will require radical treatment, while the effort will be made to save the remainder. In those mouths in which palliative or radical treatment is indicated for a number of teeth, the limit of care in preventive treatment should be applied to those teeth which have not as yet become involved in peridental disease.

Nowhere in the practice of dentistry is there better opportunity for the exercise of good judgment, based on experience and carefully recorded observation, than in determining the course to be pursued in the management of these cases. The retention of teeth which should be extracted often interferes with the effectiveness of palliative and preventive measures and the denture will be lost much earlier than it would have been had such teeth been removed in the beginning. It is especially urged that a plan must be determined upon by which the particular mouth will be kept free, or practically free, from suppuration. As the care by the patient is an important part of palliative treatment, the failure to interest and train the patient will mean that the teeth will be lost much earlier than in a similar case in which the fullest coöperation of the patient is secured. If a case presents with but one or two bad pockets and there is the opportunity to free the mouth of disease by extracting these teeth, it will often be the best course to pursue.

In all of this, it should be kept in mind that the sooner the teeth are removed, the better will be the residual alveolar ridge for artificial dentures. Therefore, we have to consider not only the present value of the teeth in mastication and the effect of the mouth condition on the general health, but also the future possibilities of mastication with artificial dentures in relation to the physical welfare of the patient.

SENTIMENT IN ITS RELATION TO THE TREATMENT OF DISEASES OF THE PERIDENTAL MEMBRANE.

There is a widespread sentiment among the laity that these diseases are somehow curable, and a search is continually going on to find practitioners who can succeed in this. We can not treat this sentiment with contempt; it is well fixed, and we must deal with it as we find it. The choice of a course to pursue is often difficult. We must contrive to convince these patients

without using means which are too harsh, and this is a most serious phase of the situation.

I may refer to a comparatively recent case which was sent to me for examination after the dentist had failed in his effort to control the discharge of pus. The patient, and several dentists who were present, were evidently dissatisfied with my decision that the teeth should be extracted. I looked over the case a little hurriedly, and found such deep pockets about so many of the teeth that there was no question as to the indication for their removal, and I advised that all of the teeth — thirty-two of them — be extracted. After I had returned to my study, one of the demonstrators came to me and said that there was a considerable objection to the carrying out of my order, so I immediately returned to the case. I had previously said nothing about having radiographs made, for the reason that in my judgment it was unnecessary. It then occurred to me that radiographs were desirable in this case from the educational point of view, and the patient was at once sent to the radiographer for this purpose. They showed extensive absorption of the alveolar process of practically every tooth. When these were studied by the patient and by the others who objected to the removal of the teeth, a complete change of sentiment occurred and the teeth were extracted. Many of the teeth, which made such a good appearance, had less than a quarter of an inch of bony alveolar process about the ends of their roots, and the incisors had but one-eighth inch of bony process remaining. We should use every means of this kind to satisfy the sentiment of our patients, for they should be positively convinced that the treatment proposed is correct, especially when we have determined that the most radical treatment must be employed. It would seem that no further argument would be necessary to convince a person that the teeth should be removed, if radiographs such as are shown in Figures 234 or 261 were exhibited and explained in comparison with others showing the normal condition of the bone, yet some will, against all advice, insist on retaining such teeth at all hazards.

The plan of examination here given is much more thorough than that which has generally prevailed heretofore. It is essential that such a close and careful examination of cases of this kind be made. This information is necessary as a basis for proper treatment. It is also necessary as a means of so presenting the difficulties to patients as to convince them that the proposed treatment is right, in order that they will agree to have

it done. In a considerable percentage of cases, the rigid search for information, and judicious care in the presentation of the evidence, will be required to put aside this sentiment and give the dentist an opportunity to apply the treatment best calculated to benefit the patient. A plain and fair presentation of the conditions found, and the treatment proposed, will usually be best.

The widespread interest of the medical profession in mouth infections in relation to general health, and the wonderful advance in popular knowledge of these things during recent years, have done much toward influencing the sentiment of our people. This will be of material assistance to the intelligent dentist in the management of his patients. It also places upon him a greater responsibility, and he should not only make the most careful study of each case, but critically observe the progress of all cases, whenever the opportunity presents, in order that his judgment may become better and better as time passes.

Abandonment of antiseptics in the treatment of suppurative pericementitis.

As for other conditions and diseases, the pathology of which has not been understood, the treatment of suppurative pericementitis has included a wide variety of medicaments. Almost every drug which might be classed as a caustic, germicide, antiseptic or sedative has been used.

The first efforts to treat these pockets were made at the time when the use of antiseptics in the treatment of infections was prominently before the medical profession. Dentists therefore undertook to control the discharge of pus by the use of the various germicides and antiseptics which were available. Various preparations of phenol, copper sulphate, zinc chloride, iodin, oil of cassia, oil of cloves, many other of the essential oils, and similar drugs were commonly used for this purpose. Many of these were caustic as well as antiseptic, and not only destroyed the organisms within the pocket, but much of the soft tissue as well, and many pockets were doubtless made deeper by the use of such drugs.

Clinical observation of the results led the more careful men to gradually change from the stronger to the milder antiseptics. Laboratory tests of the potency of the various antiseptics, also experiments showing their action on animal tissue, led to greater and greater caution in their use.

I remember the case of a young woman in whose mouth there were two very bad pus pockets. One of these was on the labial surface of the root of an upper lateral incisor, and the other on the buccal surface of the upper first bicuspid on the opposite side of the mouth. I had her come for treatment every day for some time. That was many years ago, and I undertook to stop the pus formation by using a twenty per cent solution of phenol. Under this treatment the case seemed to become worse instead of better. Then I tried ninety-five per cent phenol, using it very cautiously by wrapping a few fibers of cotton on a point and carrying that up under the free gingivæ into the pocket, being as careful as possible about injury to healthy tissue. With this the pus became less, but the pockets did not heal. Then I left off the treatment for a time, and the discharge became worse again. Then I tried pretty much everything in my medicine case — iodin, zinc chlorid, oil of cassia, and various other poisons to micro-organisms and to healthy tissue. It was of no avail. Again I allowed the case to rest without treatment. The pockets seemed to do better, but pus appeared on pressure over the roots of the teeth. I threatened to remove the soft tissue covering together with all of the diseased part, but the patient showed the teeth and gums very prominently, and this would, especially in the case of the lateral incisor, make a permanent blemish which would be almost as bad as the loss of the tooth. It was not done. Some years later both teeth were lost. In this mouth no other teeth were attacked. The teeth and gingivæ remained healthy while I knew her.

A similar case occurred a number of years afterward in which only an upper lateral incisor was affected. This case was treated by simply washing with water in which two or three drops of oil of cloves were shaken in a quarter of a tumblerful. The labial gingivæ dropped down, forming a notch in the crest line, but there was no further discharge. This case healed, not by a reattachment of the soft tissue to the cementum, but by the reduction in depth of the pocket as a result of the recession of the gingiva. Such cases are not very frequent, but I have had enough of them to make an interesting group. In more recent years the pockets in such locations which were shallow when I found them, got well under careful washing with water or salt solution. There was generally a deformity of the crest line of the gingivæ after healing, marking the spot. The deeper ones did not get well.

It should now be recognized that practically every drug

that we have used in these pockets for the purpose of destroying the micro-organisms has, in the long run, done more harm than good. Many have been successful in that they have destroyed the micro-organisms which were in the pocket, and also in that they relieved the pain, so that cases were apparently improved. We know now, however, that the drugs which destroyed the micro-organisms were also injurious to the tissues with which they came in contact; that the relief from pain was often due to the anesthetic effect of the drug; that the resistance of these tissues was so weakened by the drug that they could not for days, possibly, exercise their normal defensive activity; that they were therefore unable to meet new infections as aggressively as before; that cases, under such treatment, very gradually went from bad to worse as a result of infections.

Defense by the tissues.

To-day the indication is not so much for a drug which will destroy the micro-organisms, but rather to mechanically remove the bulk of these and keep the tissues in such condition that they will exercise their defensive powers to the utmost. This is best accomplished by a method of cleaning which will free the pocket of all organisms that may be washed out without interfering with the normal aggressiveness of the tissue cells; so that the tissues may have the best opportunity to destroy the organisms which have entered them.

The defense by the tissues themselves should be reckoned with in any treatment of suppuration. This is especially true when the suppuration occurs in the mouth. Under the conditions found in the mouth it is impossible to prevent reinfection, because of the abundance of the saliva, and its constant state of infection. Such a thing as actual surgical cleanliness can not be maintained. If the use of antiseptics upon the soft tissues of the mouth reduces the normal defense set up by the tissues themselves, and my personal observation, as related, teaches me that they do, they should not be used upon these soft tissues when these are suffering from injury or disease. Here any injury to this normal defense in the treatment is a wrong of such importance that its consideration can not be omitted. Other forms of treatment must be adopted that will be free from this objection.

Treatment by cleanliness.

Treatment by cleanliness means treatment by methodical irrigation of the wound with sterile water, or with physiological

salt solution. In all such cases there is more or less fluid — serum or pus, or both — containing micro-organisms, and the neighboring soft tissue is attacked by these organisms, which penetrate within it to variable depths. The thorough irrigation of such an infected wound or cavity removes at once the bulk of the organisms and other material which are injurious to the tissues. If plain water or salt solution is used for this purpose, there is no interference with the most aggressive action of the tissue in attacking those organisms which have entered it. On the other hand, if a strong antiseptic is used, the cavity can not be more effectively freed of its fluid contents than with water, and the medicament will, to a greater or less degree, inhibit the normal defense of the tissue.

The statement of the case may be put in this form. If one can prevent the accumulation of micro-organisms upon the tissues or in pockets about the teeth, where there is the constant tendency to reinfection, the tissue cells will be kept in a more normal state of activity, and will be able to resist the micro-organisms already in the tissues by vigorous phagocytic action. If the tissue cells are not weakened by macerating solutions, or poisoned by antiseptics, and a high degree of cleanliness is maintained, other things being equal, the quicker will the organisms which have entered the tissues be destroyed. It is for this reason that the frequent washing of pockets with physiological salt solution should be substituted for the periodical application of antiseptics. If patients can be trained to effectively wash out these pockets two or three times daily, as a part of their habitual routine cleaning, this would seem to be, and in years of trial has proven, the very best plan of treatment.

The use of physiological salt solution is recommended for all open wounds in preference to plain water, because no osmosis occurs and pain is thus avoided. If an open wound is flooded with sterile water, more or less pain is caused; on the other hand, if the irrigating fluid is of a higher specific gravity — contains more salts than the tissues, pain will result. The solution should be one that will not materially disturb the balance of osmosis between itself and the tissues in either direction. A solution of .6 of one per cent to .9 of one per cent will be very satisfactory. Such a solution may be conveniently made by adding a teaspoonful of table salt to a pint of water and sterilizing, or by the use of the specially prepared sodium chloride tablets in sterile water.

Perhaps one of the strongest illustrations of this will be

found in placing water in the eye. Pure water will be painful to the eye. A properly gaged salt solution will not be painful, and the eye may be washed in all of its parts and will continue to feel comfortable. If the salt is excessive in the solution, again the eye will be painful. Therefore it is the balance of the specific gravity of the salt solution with the blood serum and the serum in the tissues, that is necessary for painless irrigation. If the solution used is at the body temperature, this will be a further aid in the same direction.

This treatment is exactly in line with that employed by many surgeons of to-day in the treatment of infected wounds. Our use of medicaments in the treatment of these pockets has all along followed more or less closely the use of similar germicides and antiseptics by physicians and surgeons in the treatment of infections elsewhere. The time has arrived when we should abandon the use of drugs of this kind for the purposes which have been mentioned.

The following quotation from L. R. G. Crandon's work on "Surgical After-Treatment" is perhaps as fair a statement of the present position of our most progressive surgeons in regard to the use of antiseptics:

"The attempt to destroy all the bacteria in the focus by means of antiseptics is futile. That it has been a failure is attested by the fact that for the last few years the practice has gradually gravitated toward the use of extremely mild antiseptics for surgical dressings, soaks, and irrigation, such as weak boric acid, chlorinated soda, or normal salt solution. If the antiseptic solution is sufficiently strong to kill the bacteria, it will be equally efficient in its injury to tissue cells. Further, excepting in unusual cases, the antiseptic application can not be expected to come into contact with all the bacteria. Those which have escaped its action will find a good culture medium for further growth in the cells that have been injured, and in the exudation which the irritation of the antiseptic will have produced. Gauze drains have their part in making matters worse, when they obstruct the discharge and lead to the accumulation of pus and bacteria under some slight pressure.

"The persistence of infectious disease in spite of surgical effort attests in such cases surgical failure. Extirpation that does not completely extirpate, drainage that does not effectually drain, and impossible methods of destroying bacteria in the infected foci, should not be expected to lead to any but a considerable percentage of failures, and suggests the advantage of

methods that will be more effectual in the accomplishment of cure than those in present use in the treatment of localized infections."

As a proper basis for this change, we have only to review the history of the use of antiseptics in surgical operations. Such a review, if given in detail, would require a book in itself. I shall, therefore, in the following pages give an outline of the more important events, to which will be appended a list of the principal facts and discoveries along this line during the past one hundred years.

THE DEVELOPMENT OF ANTISEPTIC AND ASEPTIC SURGERY AND THE USE OF ANTISEPTICS.

ILLUSTRATIONS: FIGURES 294-295.

As one reviews the development of modern surgery, there would seem to be a natural division into three principal periods. The first may be called the preparatory period, during which a number of isolated facts were discovered, which were later brought together to substantiate the principle of the bacteriology of infection. The second should be called the antiseptic period, during which the dominant idea was the employment of antiseptics to combat and destroy pathogenic organisms. The third should be called the aseptic period, during which the principal effort was to prevent and combat infections by aseptic methods. If, however, one endeavors to actually separate the steps of progress into these three periods, it will be found quite impossible to do so. It will then be recognized that the development has been very gradual, yet continuous, without sharp divisions, from the original announcements of Lister in 1867, to the present day. In fact, many discoveries previous to the time of Lister were necessary to this development, and some of these formed the foundation for the work of Lister and those who followed him.

Preparatory period.

The perfecting of the achromatic microscope by J. J. Lister in 1830 made possible the description of epithelial tissue in animals by Henle in 1837, and of plant cells by Schleiden in 1838. These were preliminary to the development of Schwann's cell theory in 1839. Holmes pointed out the contagiousness of puerperal fever in 1843 and Semmelweiss discovered its cause in 1847. Cohn pointed out the vegetable nature* of bacteria in 1853, and Pasteur's investigations of the chemical products of putrefaction in 1856, led up to the discovery that bacteria were the cause of putrefaction. The publication by Virchow in 1858 gave to the world a new understanding of the cellular structure

* Recently Vaughn (Journal American Medical Association, August 1, 1914) and Kossel have shown "that bacteria contained no cellulose and are particulate, unshielded proteins, and consequently more nearly related to low forms of animal life." While Vaughn says he would not classify bacteria as either plants or animals, in their life processes they are more closely related to animal life.

of animals and plants, and of the nature of disease processes. A little later, in 1860, Lemaire pointed out the antiseptic properties of carbolic acid (phenol). Pasteur had shown in 1861 that the fermentations were caused by micro-organisms and had put the evidence of this in more definite shape in 1866. In the meantime Lister had become profoundly impressed with the communicability of erysipelas, gangrene and septicemia, and had arrived at the conclusion that if micro-organisms were the cause of fermentations occurring outside the body, as had been demonstrated by Pasteur, these communicable diseases might be the result of fermentations in the body tissues. He also reasoned that, if he could prevent the entrance of micro-organisms into the tissues, or destroy those that had entered, he could prevent or cure the diseases.

Antiseptic period.

When Lister reported in 1867 his wonderful experiments of the preceding years in operating under a spray of phenol and the securing, in cases of deliberate surgery, of wounds free from infection, which healed without pus, his announcement of these facts shook the very foundations of the old-time surgery. At that time suppuration was considered to be a necessary part of the healing in all considerable wounds. Lister had followed the controversy between Baron Leibig, the great German chemist, and M. Louis Pasteur, the French chemist, in which Pasteur proved step by step that fermentation in wines, vinegars and other fluids was not due to a chemical change produced by "molecular motion," as contended by Leibig, but was due to the growth in such fluids of minute organisms or germs. In making this proof the chemist Pasteur developed a new and revolutionary science — the science of bacteriology — and became the first and the greatest bacteriologist. Lister followed this proof of the role of germs in producing fermentation and concluded that germs probably played a similar role in the body in producing septicemia and other surgical diseases. What Lister really thought to do was to prevent, if possible, the decomposition of pus in operative wounds. For it was to this, and not to laudable pus — as a clean, pure pus was then called — that surgical fever, which was destroying the lives of so many surgical patients, was attributed. The prevention of pus formation had not had a place in his thought. The result was as much a surprise to Lister as to others.

While to Lister is undoubtedly due the credit for develop-

ing antiseptic surgery, it should be recognized that he undertook this study as a result of the work of Pasteur, as is shown by the following quotation from a paper* by Lister in 1873. "The philosophical investigations of Pasteur long since made me a convert to the germ theory, and it was on the basis of that theory that I founded the antiseptic treatment of wounds in surgery."

Thus antiseptic surgery was born, and from 1867 to this date the belief in the truth of Lister's deductions and the development of methods for perfecting details for efficiency have never ceased. No sooner had the fundamental discoveries of Pasteur and their practical application to surgery by Lister begun to be accepted by the profession than hundreds of men in every country took up investigations which supplemented and expanded the new science and art of surgery. Abbè introduced the oil immersion lens in 1872. Methods of staining bacteria were perfected by Weigert in 1871, by Ehrlich in 1874, by Koch in 1876, and many others, thus preparing the way for the isolation and recognition of the various pathogenic bacteria. In the meantime the antiseptic properties of numerous drugs were demonstrated. This was accomplished by application of the antiseptics to wounds to destroy micro-organisms that had entered them, which was followed by methods of dressing, devised to prevent the re-entrance of micro-organisms into these wounds during the healing process. The antiseptics were also used to prevent infection of food and water in sanitation and in hygiene.

Lister's first experiments consisted of the employment of assistants who stood near by and kept the atmosphere and everything in contact with the wound sprayed with a phenol solution, using hand atomizers for the purpose. From this beginning, every conceivable form of experiment was tried. As these studies progressed, it was found that the formation of pus was caused by the ingress of certain micro-organisms, comprising only a few species, which seemed to be common throughout the world, and were called pyogenic organisms, that is, pus producers. Pasteur isolated the streptococci and staphylococci in 1880.

Strong antiseptics, principally phenol solutions in the

* "A Contribution to the Germ Theory of Putrefaction and Other Fermentative Changes, and to the Natural History of Torulæ and Bacteria." A communication to the Royal Society of Edinburgh in 1873, published in the Transactions of the Royal Society of Edinburgh, 1875.

beginning, were used to antisepticize everything which might come in contact with the field of operation. After a time it was learned that the spray of phenol was unnecessary because other antiseptic precautions proved sufficient. However, for probably fully twenty years after Lister's report in 1867 the view among physicians and surgeons that micro-organisms from the air infected wounds, was quite general. The sponges of earlier days were discarded for cotton rolls and gauze, which could be made safe by antiseptics. Instruments were immersed in antiseptics, the tissues within the field of operation were saturated with antiseptics; the hands of the operator and his assistants were scrubbed and then bathed with antiseptics, many operators immersing their hands in two or more different solutions. All wounds were irrigated with antiseptic solutions. This was what has come to be known as the antiseptic period in surgery.

One of my professional associates, Dr. David Prince, visited Lister's clinic as well as other clinics in Europe, and on returning to Jacksonville, Illinois, equipped an operating-room with an elaborate system of fans, sprays and filters "for securing an aseptic operating-room." I have had reproduced an illustration of this operating-room, taken from a paper entitled: "The Relation of Micro-organisms to Aseptic Surgery," by Carl E. Black, M.D., published in 1887. A brief description accompanies the illustration. (See Figure 294.)

Aseptic period.

As the facts regarding infected wounds became established, it opened the way to the recognition of sterile wounds as something distinct from infected wounds, and led to the treatment of these by methods of dressing that would exclude micro-organisms during treatment, which was demonstrated to be sufficient. This was the beginning of the aseptic period. In deliberate operations, aseptic methods gradually replaced antiseptic methods. After a time it came to be realized that a deliberate operation might be performed under such perfect conditions of asepsis that from the beginning of the operation until the wound was entirely healed, the tissues could be kept practically free from micro-organisms, and when this could be done antiseptics were unnecessary. This came to be known as surgical cleanliness, which means freedom from micro-organisms.

Use of antiseptics gradually lessened.

Very gradually the ideas of the medical world have been changing and leading away from the use of antiseptics on the

soft tissues in the treatment of infected wounds, because the development of more perfect cleanliness pointed to a better way. The application of the principles of surgical cleanliness to infected wounds is leading to the gradual abandonment of the use of antiseptics in contact with the tissues.

The first idea of an antiseptic was that of a drug which would destroy micro-organisms, but would not injure the animal cell. The search for such a drug failed. The next idea of an antiseptic was that of a drug which would stop the growth of micro-organisms without very material injury to the animal cell. Many drugs are used to-day with the idea that they will do this. This is an error. It seems that such a drug does not exist to-day. There are drugs which will temporarily stop the growth of micro-organisms without actually destroying the animal tissue, but they inhibit, or tend to inhibit, the activity of the animal cells, and the repair of the wound is delayed for a time. This has been well tried out, both on animals and human subjects.

One of the greatest disappointments in Dr. Koch's life was his failure to cure tuberculosis by the use of antiseptics. He could sterilize the cages of his animals and give them clean food, and prevent them from taking tuberculosis, but if one took the disease through any mismanagement, or was purposely infected, he could not cure it. The greatest disappointment in the life of Dr. Miller occurred after he had determined the cause of decay of dentin. He fondly hoped to prevent caries by finding an antiseptic with which he could sterilize the human mouth. In this he failed. He could not sterilize his own mouth for a single hour with any drug which he could use. He could greatly reduce the number of organisms in his saliva by careful washing with drugs, or with plain water, but in a few hours they would be as plentiful as ever. I have done similar experimental work, and met with the same disappointments.

Chemotaxis and phagocytosis.

There are some theories hinging around chemotaxis, positive and negative, which serve to explain what really happens, whether the theories are correct or not. Certain micro-organisms are picked up and destroyed by the phagocytes. I have seen and studied the cells at this work, resupplying the examples as the first became still, watching them for hours at a time on the warmed stage of the microscope in inflamed tissue now and then clipped for the purpose. The phagocytes will approach and pick up certain micro-organisms. Other certain

micro-organisms they will run away from. In the one case we have chemical conditions which bring the phagocytes into the field — positive chemotaxis. In the other case the micro-organisms produce chemical compounds which cause the phagocytes to run away — negative chemotaxis.

Metchnikoff and some of his brilliant pupils at the Pasteur Institute in Paris developed the study of phagocytosis. He divided phagocytes into "macrophages" or "fixed phagocytes" and "microphages" or "wandering phagocytes." Macrophages include large lympocytes, myelocytes, giant cells and other cells which do not form a distinct group, but are known by the size of the cells and seem to have the function of digesting tissue which is moribund or dead. On the other hand the "microphages," which are "practically identical with the neutrophile and eosinophile polymorphonuclear leucocytes, have the function of taking up bacteria and digesting them — bacterial phagocytosis."

Conditions similar to chemotaxis may be produced by the action of drugs. Most antiseptics induce a negative chemotaxis which drives the phagocytes out of the field until the effect has passed. During this time the healing process and the formation of pus will be suspended. Both will be renewed as the drug disappears from the tissue.

It should be borne in mind that Vaughn and others have proved that all cells in the body, no matter of what tissue, have the power to take food (protein) from the blood and lymph streams. Sometimes, especially in the presence of disease — "an inflammatory process from infection and the efforts at repair" — the cells take up food which is injurious to them. In fact such cell injury is disease.

Personal studies.

Soon after the publication of Lister's reports, I became interested in this whole field of investigation. I soon had a very complete laboratory equipment and was making cultures and studying the various micro-organisms at first hand from all kinds of abscesses, carbuncles and suppurating wounds, so that I became quite familiar with them. I watched surgeons closely, saw much of surgical cases, and did some operating myself. I read a number of papers before medical and dental societies, and in 1884 published a small book entitled "The Formation of Poisons by Micro-organisms." Subsequently I made extensive studies of the potency of the various antiseptics in use by sur-

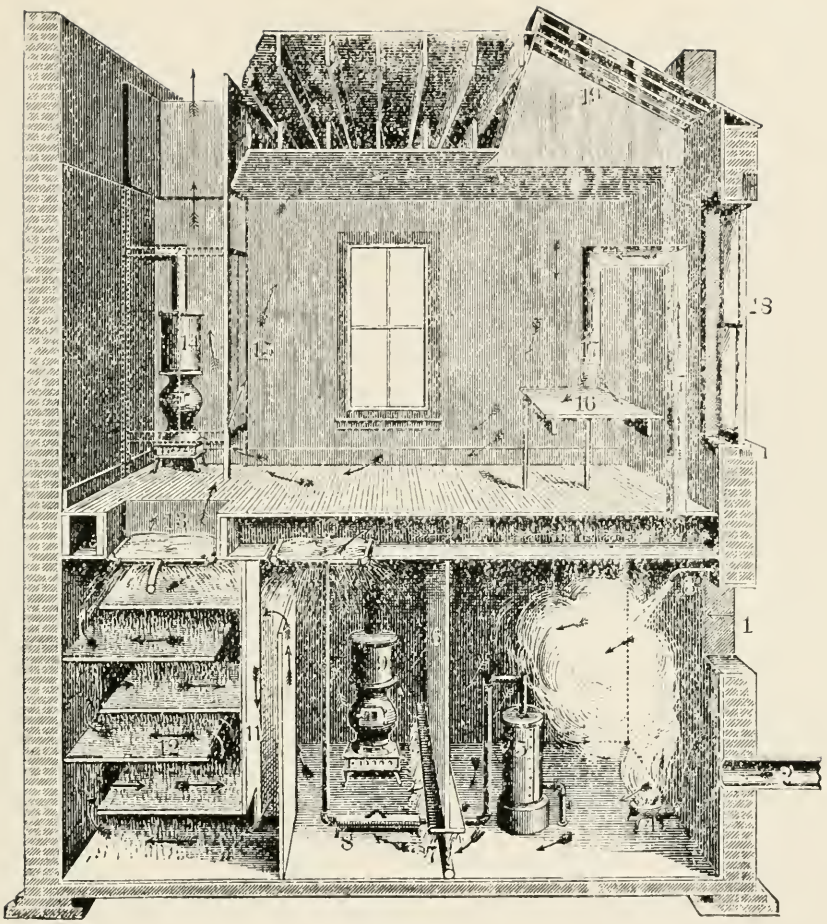

Fig. 294.

Fig. 294. Surgical operating room equipped by Dr. David Prince, Jacksonville, Ill., in 1887. The following description is quoted from the article by Dr. Carl E. Black, referred to in the text:

"The room is a separate building connected with the hospital building by a porch. All the air admitted to the operating room above comes from the basement thoroughly washed. The air passes into the basement at 2, through a steam jet 3, into a chamber where sulphur is constantly burning, 4. It then passes under a muslin diaphragm 6, through sprays of cold water 7, into a second chamber which is warmed by a stove 9. It next passes between a series of muslin diaphragms 11, 12, which are kept dripping with cold water from a rosette 13, and is then in the operating room. Ventilation is effected through a flue 14. Thus all the air entering this room is thoroughly sterilized and the operation is carried on in an aseptic atmosphere. This room contains only such things as are necessary during operations. There are no closets, and the floors, besides being of several layers, one of which is tarred paper, is filled with paraffin. A north light is secured through a window 18, and a skylight 19."

Fig. 295.

Fig. 295. Radiograph taken in 1914 of the elbow of arm injured by shotgun in 1878. The treatment of this arm by keeping it immersed in a phenol solution bath for six weeks is described in the text. There was no infection, neither was there any formation of granulations, while the arm was immersed. The action of the antiseptic on both the micro-organisms and animal tissue was well demonstrated by this case.

geons, physicians and dentists, and the effect of these on animal tissue. In 1886 and 1887 I gave a series of lectures and demonstrations on this work before the Illinois State Dental Society.* My ovens have been in practically continuous operation up to the present time. In the consideration of the use of drugs in the treatment of the dental pulp, reference is made to a very interesting report of an investigation into the effect upon animal tissue of the various antiseptics used in dentistry, made in my laboratory by Dr. A. H. Peck in 1897. There is also a brief report of recent similar experiments, some of which are illustrated in Figures 347 to 358.

It was laboratory work of this kind, by many men in the medical and dental professions, which, together with clinical observations and practical surgical demonstrations, led to the changes in the use of antiseptics which have been mentioned.

One of the most marked experiments showing the effect of carefully used phenol upon the animal tissue occurred under my personal observation and management. This was in 1878, when there was a flush of excitement over some reports of advantage in keeping wounds immersed in water with very moderate use of antiseptics during the earlier part of the treatment.

A boy of fourteen, while hunting, accidentally discharged his shotgun in getting over a fence. The charge struck the ulna of the right arm two inches above the wrist, went through it diagonally, and left it about two inches below the elbow joint. Examination for shot and finding many in the tissues about the elbow, indicated that there were enough shot in the joint to destroy its usefulness. The radius was uninjured, the radial artery was in good condition and the circulation in the hand was good. The hand would live and might be valuable if that terrific wound could be managed. The whole of the ulna between the points named was gone, and much of the soft tissue was shot away. The wound lay wide open for the whole distance. An apparatus that answered the purpose of holding the temperature of the water very close to the body temperature and kept it in continuous circulation, was quickly made and installed. This included a pan, or trough, in which the arm could be laid and swung above the bottom of the pan. The wounded arm was laid in it within three hours after the injury. The entire wound was submerged. The tissues were formed up a bit, but they were not brought together. All parts could be inspected and a

* Transactions of the Illinois State Dental Society, 1886, p. 180; 1887, p. 162.

syringe could be used for extra washing of any particular parts. At first five per cent of phenol was added to the water. It soon became evident that this was too much, and it was reduced to two per cent. The boy soon became comfortable, or as nearly so as his confined position would permit. He found ways to make some change in position without disturbing the arm. He settled down for a long wait in bed, seemed comfortable, made no complaint of pain, spent much time in reading, was fond of seeing visitors and talking with his friends who called. Indeed this feature of the case was very remarkable.

At the end of a month no progress toward healing could be observed in the wound. Not a single point of granulation had been seen. The boy seemed perfectly well in every other way. Under these conditions the treatment was continued two weeks longer. Still there was absolutely no healing. Then the arm was removed from the bath and placed in dry dressings, no more antiseptics were used. The case was carefully watched. It was two weeks longer before the first appearance of granulation occurred. After that other points of granulation showed themselves here and there, growing very slowly at first, but increasing in vigor, and an encrustment of the tissues of the wound which had occurred in the bath, began to be shed away. Then the forming up of the tissues that remained was begun by the use of bandages. They were gradually drawn together day by day, to bring the remaining portion of the arm into the best shape possible, and to reduce the final area of cicatrization to the narrowest limits by bringing the skin that remained as closely around it as we could. Under this treatment and great care in maintaining aseptic conditions, the wound went on to cicatrization. The X-ray picture of the arm exhibited here was taken about thirty years after the accident and shows the shot still imbedded in the tissues about the elbow. (See Figure 295.)

I regard this case as the best representation of what antiseptics will do in delaying tissue growth in the healing process that I have ever witnessed. The surgeon, Dr. David Prince, was with me in the treatment of the case from the beginning to the end, and was intensely interested in every part of it. While the effort was to do the best thing possible for the boy, the whole case was also a study of the healing process under extremely difficult conditions, and especially of the effect of the antiseptic in delaying the growth of granulations, and generally delaying the normal healing processes. In this it is a good type of the effect of the antiseptics as a class, tried out to the ultimate limit.

CHRONOLOGICAL LIST OF PRINCIPAL EVENTS IN THE DEVELOPMENT OF ANTISEPTIC AND ASEPTIC SURGERY

PREPARED BY CARL E. BLACK, M.D., JACKSONVILLE, ILL.

1822. Pasteur born.
1827. Lister born.
1830. J. J. Lister perfected achromatic microscope.
1831. Liebig and others discovered chloroform.
1831. Hueter first used chlorine water and creosote as disinfectants.
1832. Liebig discovered chloral.
1836. Schwann discovered pepsin.
1837. Gerhard differentiated typhoid and typhus (clinically).
Henle described epithelial tissue.
1838. Schleiden described plant cells.
1839. Schwann published treatise on cell theory.
Purkinje first used term *protoplasm*.
1842. Long first used ether as anesthetic.
1843. Holmes first pointed out contagiousness of puerperal fever.
1844. Wells discovered nitrous oxid as anesthetic.
1845. Langenbeck detected actinomyces.
1847. Semmelweis discovered cause of puerperal fever.
Pasteur proved that fermentation was caused by microorganisms.
1850-53. Cohn declared animal and vegetable protoplasm identical.
1853. Cohn demonstrated vegetable nature of bacteria.
1854. Schroder and van Dusch proved efficiency of cotton plugs for test-tubes and flasks.
1856. Panum investigated chemical products of putrefaction.
1858. Virchow published "Cellularpathologie."
1859. Darwin's Origin of Species published.
Bataille proposed use of alcohol to promote the healing of wounds.

1860. Lemaire pointed out antiseptic properties of carbolic acid.
1861. Pasteur discovered anaërobic bacteria.
1863. Pasteur investigated silkworm disease.
1865. Villemin demonstrated infectiousness of tuberculosis.
1867. Lister introduced antiseptic surgery.
1871. Weigert stained bacteria with carmine.
1872. Abbè introduced oil immersion lenses.
1873. Obermeier discovered spirillum of relapsing fever.
1874. Ehrlich introduced dried blood smears and improved staining methods.
Hansen isolated bacteria of leprosy.
1875. Landois discovered hemolysis from transfusion of alien blood.
Losch observed parasitic amebæ in dysentery.
Lister reasoned that pus formation was caused by micro-organisms.
Thiersch proposed use of salicylic acid as an antiseptic.
Landois discovered that animal serum will hemolyze human serum.
1876. Koch grew anthrax bacilli on artificial media.
Bollinger discovered actinomycosis in cattle.
1877. Pasteur discovered bacillus of malignant edema.
Von Bergman introduced corrosive sublimate antisepsis.
1878. Koch discovered causes of traumatic infections.
Pasteur discovered micro-organisms of surgical and puerperal infections.
Israel (1845 von Langenbeck) discovered actinomycosis in man.
1879. Neisser discovered gonococcus.
Manson proved mosquito vector of filaria sanguinis hominis.
1880. Pasteur isolated streptococcus and staphylococcus.
Ponfick proved identity of human and animal actinomycosis.
Baelz discovered parasitic hemoptysis.
Pasteur established bacterial cause of lobar pneumonia.
1880. Eberth isolated typhoid bacillus.
Mosetig Moorhof introduced iodin in surgery.
1881. Laveran discovered parasite of malarial fever.
Medin discovered epidemic nature of poliomyelitis.
Koch introduced plate cultures.

Finlay first suspected mosquitoes to be carriers of yellow fever.
Reed, Carroll, Argamonte and Lazear proved mosquito to be intermediate host in yellow fever.
1882. Koch discovered tubercle bacillus.
Loeffler discovered bacillus of glanders.
Walther Fleming investigated cell division.
1883. Klebs (Edwin) and Loeffler discovered diphtheria bacillus.
Fehleisen isolated micro-organism of erysipelas.
Pasteur vaccinated against anthrax.
King first suggested that mosquito was intermediate host and sole source of infection in malaria.
Uuna introduced ichthyol.
1884. Koch discovered cholera bacillus.
Nicolaier discovered tetanus bacillus.
Finkler and Prior established bacterial cause of cholera nostras.
Crede introduced silver nitrate instillations for infantile conjunctivitis.
1885. Weismann published memoirs on continuity of the germ plasm.
Marchiafava and Celli describe hemocytozoa.
1886. Escherich discovered bacillus coli.
Von Bergman introduced steam sterilization in surgery.
Loeffler established bacterial cause of swine erysipelas.
Soxhlet introduced sterilized milk for nutrition of infants.
1887. Clark discovered bacillus of Malta fever.
Weichselbaum discovered meningococcus.
1887. Bruce established bacterial cause of Malta fever.
1888. Institute Pasteur founded.
Roux and Yersin investigated the toxins of diphtheria.
Nuttall discovered the bactericidal powers of blood-serum.
Breiger first isolated toxins.
Gamaleia established bacterial cause of fibrinous pneumonia.
1889. Buchner discovered alexins (protective bodies).
Behring discovered antitoxins.
Smith discovered parasite of Texas fever.
Buchner discovered bactericidal effect of blood-serum.
1890. Imperial Institute of Experimental Medicine founded at St. Petersburg.

Behring treated diphtheria with antitoxin.
Koch introduced tuberculin.

1891. Institute for Infectious Diseases opened at Berlin under Koch.
Lister Institute for Preventive Medicine (London) founded.

1892. Pfeiffer discovered bacterium of influenza.
Welch and Nuttall discovered bacillus ærogenes.
Maragliano, (1901) Landsteimer, (1901) Eisenberg, discovered that serum of diseased and even normal donors will hemolyze alien blood.

1893. Smith and Kilbourne demonstrate transmission of parasitic diseases by arthropoda.
Gilbert discovered paracolon and paratyphoid bacilli.

1894. Kitasato and Yersin discovered plague bacillus.

1895. Pfeiffer discovered bacteriolysis.

1896. Max Gruber and Widal discovered bacterial agglutination.
Widal and Sicard introduced agglutination test for typhoid fever.
Gilchrist discovered blastomycosis.

1897. Shiga discovered dysentery bacillus.
Bordet discovered bacterial hemolysis.

1898. Loeffler and Frosch investigated filterable viruses.
Looss demonstrated transmission of hookworm infection.
Theobald Smith differentiated between bovine and human tubercle bacilli.
Nocard & Roux established bacterial cause of bovine peripneumonia.
Schenck discovered sporo-trichosis.

1899. Reed and Carroll established transmission of yellow fever by mosquitoes.

1900. Widal and Ravant introduced cytodianosis.

1901. DeVries stated mutation theory.
Dutton and Ford discovered parasite of sleeping sickness.

1902. Firth and Horrocks showed that flies could carry typhoid bacilli.

1903. Metchnikoff inoculated higher apes with syphilis.
Bruce showed sleeping sickness to be transmissible by tsetse fly.

Richet, (1906) Rosenau and Anderson, (1907) von Pirquet investigated Anaphylaxis which was discovered by (1798) Jenner and (1839) Magendie.

1905. Schaudinn discovered parasite of syphilis.
Bordet and Gengou discovered bacillus of whooping-cough.

1907. Wasserman introduced sero-diagnosis of syphilis.
Von Pirquet introduced cutaneous reaction in tuberculosis.

1909. Ehrlich introduced salvarsan.
Hunter introduced the term "Oral Sepsis."
Nogouchi improved the Wasserman reaction.

1910. Henri and others introduced ultraviolet sterilization of water.
Flexner produced poliomyelitis experimentally.

1911. Nogouchi introduced luetin reaction.
Peytron Rous transmitted sarcoma by means of a filterable virus.

1912. Bass cultivated malarial plasmodium in vitro.

1913. Abderhalden introduced ferment reaction for diagnosis of pregnancy and dementia praecox.
Vaughn and Kossel showed bacteria contained no cellulose and are more nearly related to animal than vegetable life.

ACUTE ULCEROUS GINGIVITIS

ILLUSTRATION: FIGURE 296.

A disease which I have noted as occurring rather infrequently has been described by Dr. Thomas L. Gilmer as *Acute Ulcerous Gingivitis* — a term which seems to me to be apt. This form of gingivitis stands entirely apart from the diseases of the gingivæ and peridental membrane which have been described. For this reason, the description of it was not placed with the other forms of gingivitis, in order that confusion might be avoided. Although I had seen the disease previously, I had not written anything regarding it for publication, and Dr. Gilmer's description* is the first that I remember to have seen. It is so accurate a description that I quote it here in full.

"Acute ulcerous gingivitis is a disease seen but rarely and has not, so far as I am able to discover, been previously described. The onset of the disease is sudden, the earliest symptoms indicated by a slight malaise which is quickly followed by rapid ulceration, at first confined to the gingivæ, usually about two or three of the anterior teeth on both jaws simultaneously and in corresponding localities; later it is extended to the gums about a number of the teeth, or groups of teeth, but rarely if ever does it include the entire gum margin. The lingual margins and festoons of the gums do not participate at first in the inflammatory processes, but later the festoons are destroyed and deep pockets are formed in the interproximal spaces. Still later the lingual gingivæ participate, but in no case have I seen ulcerous manifestations in this locality. In twenty-four hours after the patient's attention has been called to the condition of his gums, the parts attacked present the appearance of having been gnawed away until most of the gum tissue overlying the alveolar process immediately adjoining the teeth has been destroyed. The part of the gum attacked has soft, thickened, and in some instances everted margins. The eroded parts form pockets which are filled in with a grayish pasty mass, similar to that found in syphilitic ulcers in the mouth. When this mass is removed a granulation surface is exposed which bleeds easily and is very painful to the touch. The mucosa for a short dis-

* Dental Review, Vol. 20, 1906, p. 459.

tance from the ulcerative margins is of a dark red hue as a result of the congestion. The gum covering the cervical and interproximal surfaces of the teeth is destroyed sufficiently in some cases to uncover the teeth down to the alveolar border.

"The breath of the patient is fetid, the saliva ropy and in excess of the normal. The temperature in the early stages of the disease ranges about 101 degrees. The patient is nervous and anxious, the appetite is poor, sleep is disturbed, the bowels are constipated. Bloody saliva escapes from the mouth during sleep, staining the pillow.

"Without treatment the ulceration does not appear to extend beyond the gingival border of the gums, nor deeper than the alveolar process, but when the case is neglected the entire mucosa of the mouth in a degree participates in the disease, that is, to the extent of becoming red and tender. The ulceration seems to be limited to the areas indicated, and after the destruction of the mature tissue has ceased, the granulations only appear to be destroyed, leaving the pasty mass covering.

"The submaxillary lymphatics become involved early in the disease. Microscopically no pus is observed, but the microscope shows pus cells in abundance.

"It is not necessarily a disease of the poor, or those of impaired vitality. It is not confined to those who are careless of oral hygiene; neither is it traceable to metallic poisons, syphilis or renal disease.

"With treatment, consisting of local applications of antiseptic washes only, the disease yields slowly, extending over a period of two weeks or more.

"After having treated a few cases by local antiseptic applications only, and finding the obstinacy of the disease to such treatment, and knowing how sensitive the gums are to mercury, I concluded to try its effect by giving small doses of calomel in combination with the local treatment, with the most happy results. The home treatment consists of taking four tablets of mercurous chloride, 4/20 grain each, morning, noon and night, followed by a saline cathartic on rising. The gums are thoroughly cleansed before breakfast by a swab of cotton dipped in three per cent pyrozone. Every two hours during the day the mouth is flushed with a warm saturated solution of boric acid, to which is added one drop of the oil of cassia to two ounces of the solution. The office treatment consists in a thorough cleaning of the parts in the afternoon with three per cent pyrozone, followed by the application of compound tincture of benzoin.

On the third day under this treatment a marked improvement has uniformly been observed, and in a few days more the gums have been fully restored. In the place of the benzoin, dilute tincture of iodine may be substituted with equally good results.

"I have twice attempted through cultures to discover a specific organism to which the disease might be attributed, but without success. When another case presents further search will be made for the cause."

The cases I have observed have been substantially like those described by Dr. Gilmer; some have been much worse, not only destroying the gingivæ to the margins of the alveolar process, but uncovering the alveolar process and producing exfoliation of it from the roots of the teeth for nearly half their length. Some of the cases have been much milder and have produced only the sloughing of the free gingivæ. They have all been attended with the general symptoms described by Dr. Gilmer, and in some of them the swelling of the lymphatics about the neck was very great, but so far as I remember, none of these suppurated.

It is not characterized by large amounts of pus, but there is a pasty mass that seems to confine and cover over the micro-organisms which appear to be instrumental in causing the disease. Like Dr. Gilmer, I have searched for the specific micro-organism, but also like him, I have failed to discover such.

Most of my cases have been seen in persons of mature age, but a quite notable one occurred in a child about nine years old. This case was brought in to me by a dentist. It was a severe case, the sloughing occurring both in the upper and lower jaws in the positions mentioned by Dr. Gilmer.

The first of these cases which presented to me was treated with local antiseptics after a somewhat vigorous saline cathartic. With this treatment I have had the same results as described by Dr. Gilmer. After studying some of these cases and especially the characteristic viscid covering which overspread the diseased area, I decided to try another method. I used salt solution only and persisted in the washing until I had thoroughly cleaned the tissues of everything that could be washed away. This is no easy task, and it is rather painful to the patient, for it requires considerable force in the current of water thrown. I have taken the ordinary two-ounce rubber bulb syringe and have thrown the entire contents against the tissues with all the power that the grip of my hand could bring to bear. It has sometimes required

four or five syringefuls to clean the parts; but when once so cleaned, recovery has been rapid.

A few years ago I had a patient, quite a large, husky man, who was referred to me by his dentist. The case was in an early stage; loss of tissue by sloughing had not yet begun, but was imminent. He was nervous, so much so that I doubted whether I would be able to wash the tissues as thoroughly as I desired. I told him the treatment would be painful and used the syringe with sufficient force to stretch the tissues away from the teeth, and removed every particle of the pasty material. Afterward I told him that I should not hurt him so badly again, and used several syringefuls of water with less force, until I had the whole surface entirely clean. A saline cathartic was prescribed. This case received two additional washings and the inflammation gradually subsided. On the second day the fever had abated to very nearly normal. The case was well at the end of the fourth day. This is perhaps the most rapid recovery that I have observed.

In the case of the child previously mentioned, sloughing had already occurred, exposing the teeth down to the margin of the alveolar process in both the upper and lower jaws. The treatment of this was the same as that just described above, except that the force of the stream was moderated on account of the pain caused by it. The weaker force of the stream was compensated by the persistence of its application, and finally the granulations were laid bare at every point.

It will be seen that this treatment was on the supposition that if we could remove the micro-organisms that were growing in the pasty mass upon the surface, and prevent the continuous reinfection of the tissues, the phagocytes would quickly take care of the micro-organisms already in the tissue. To facilitate the action of the phagocytes it is necessary to keep the tissues in very good active condition, and it seems to have been successful, not only in the cases here reported, but also in several others.

I have treated but five cases by this method, all of which have done well. This number of cases is not sufficient, however, to fully establish the efficacy of the treatment.

A model of one case is shown in Figure 296. The tissues were too much inflamed to permit of an impression being taken until after the most acute stage had passed, and the illustration falls far short of conveying a good idea of the condition. The

tissues appear to be much too smooth, and there is no showing of the angry character of the inflammation. It does, however, give some idea of the extent to which the gingivæ were destroyed. In many cases the destruction is much more extensive.

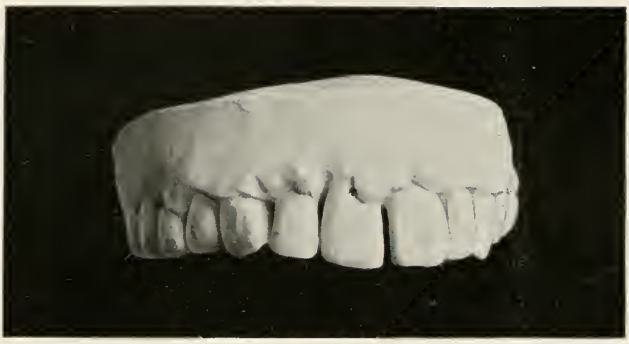

Fig. 296.

Fig. 296. Plaster model of a case of acute ulcerous gingivitis, taken after the case began to show improvement. This illustration does not give a good idea of the raggedness of the gingivæ, nor of the very angry appearance. It does, however, show something of the extent to which the tissue was destroyed.

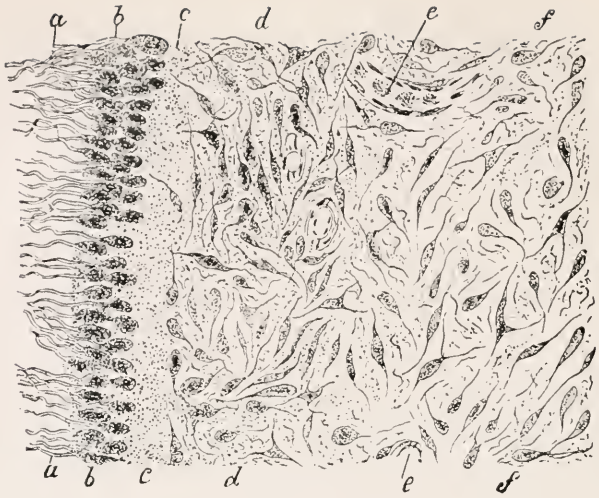

FIG. 297.

FIG. 298.

FIG. 297. Margin of the dental pulp: a, a, Dentinal fibrils, pulled out of the dentin. b, b, Layer of odontoblasts. c, c, Transparent zone between the odontoblasts and the cells of the pulp proper. d, d, Layer of cells closely packed together. e, e, Blood vessels. f, f, Cells less closely placed toward the central portions of the pulp.

FIG. 298. Odontoblasts clinging to a fragment of imperfectly developed dentin. The tissue was pulled away in mounting the section. The cells are drawn just as they lay distorted in the mounting, but a good idea is given of their true form.

THE DENTAL PULP

HISTOLOGY AND PHYSICAL FUNCTIONS

ILLUSTRATIONS: FIGURES 297-303.

THE dental pulp is that bit of soft tissue which fills the pulp chamber and root canal, or canals, within the tooth. The structure of the pulp, the more important cellular elements and their functions will be described.

THE CELLULAR ELEMENTS.

The mass of the tissue, particularly in the bulb of the pulp, is almost of a gelatinous character, very much like the gelatinous tissue of the fetus. It is therefore but a partially developed tissue. This tissue is, however, in certain parts fairly well filled with cellular elements. (See Figure 297, also Figure 35.)

In the root portion these cells are fusiform and tolerably abundant. Each cell gives off a process from each end, which rapidly becomes smaller until it is a mere thread, which can be seen only with the highest power of the microscope. These threads wind about among the cells and through the gelatinous portion of the tissue, often filling it very thickly with the minute thread-like extensions. In the bulb of the pulp, particularly of the molars, and in the larger pulps of the incisors and cuspids, the cells are more sparsely placed, and are larger. They may be round or cubical in form, each cell giving off a number of these processes which radiate in every direction through the gelatinous mass, curving here and there. The fusiform cells in the root portion are disposed with their length parallel with the length of the root canals, but in the bulbal portions of the pulps such relation is lost. In the root portion the cells are sufficiently numerous to form a fairly close tissue. In the bulbal portion they are not so plentiful.

In a tolerably thick section brought under the microscope the cellular elements will seem plentiful, even in the bulb of the pulp, but if the section be a very thin one, the cells are seen to be separated from each other to such an extent that they fail to

touch and unite with each other to form a complete cellular tissue, much of the gelatinous mass appearing as a clear space except for the windings of the very fine processes given off from the cells.

In the ordinary illustrations of pulp tissue, particularly in the photographs, little is shown of these very fine processes because they are too small for the camera to take note of them. Near the cell, a portion of the process, which is larger than the rest, is generally shown, but even this is absent from many photographs. Some idea of these processes may be obtained from Figure 297.

The odontoblasts. Fibrils of Tomes.

All around the periphery of the pulp there is a zone in which the cellular elements are more closely placed, forming what would seem to be a higher grade of connective tissue, but even this is very imperfect. Upon the surface of the pulp tissue, between it and the wall of the dentin enclosing it, there is a layer of small elongated cells, which, when cut through perpendicularly with their length, look like a layer of fine columnar epithelium. These cells are called *odontoblasts* and their principal functions are the building of the dentin, the maintenance of its vitality, and the transmission of the sense of pain. They also have what seems to be the function of protecting the pulp from exposure by building secondary dentin in response to certain stimuli. This will be discussed in the consideration of calcifications within the pulp chamber.

The odontoblasts are very closely placed. (See Figures 297, 298 and 299.) Each of these cells gives off a process which enters one of the dentinal tubules and passes from the pulp through the dentin, usually in a somewhat curved direction, and ends at the dento-enamel junction in the crown portion, or at the dento-cemental junction in the root portion. These are the *fibrils of Tomes,* or the dentinal fibrils discovered by John Tomes, about 1840. These fibrils, which radiate through the dentin in every direction, give sensitiveness to this tissue. Indeed, each of these fibrils seems to be an extended portion of the odontoblast. (See Figures 297, 302 and 303.) The odontoblast itself is apparently a very sensitive element of the pulp tissue, and by means of the fibrils, conveys that sensitiveness from all parts of the dentin.

Blood vessels.

The pulp tissue has a fairly abundant circulation of blood,

conveyed to it by one or more arteries, accompanied by returning veins, which pass into it through the apical foramen in the fully developed tooth. (See Figure 300.) In the formative stage of the tooth the blood supply is much more abundant, the tissue of the future pulp having a broad base through which many arteries enter. As the growth continues and the pulp is narrowed down to adult dimensions of the pulp chamber, this blood supply is cut off more and more, until often there is but a single minute artery entering the tissue, accompanied by a returning vein. This artery begins to break up in the canal portion of the pulp, sending branches to all parts, and this is continued even in the bulbal portion, until it is divided into fine arterioles, which approach the layer of odontoblasts previously mentioned. These again divide into a plexus of capillaries, which are especially abundant near the pulpal ends of the odontoblastic layer. The blood supply continues to be fairly rich in the pulp of the adult tooth.

Walls of the blood vessels. The walls of the blood vessels of the pulp are unusually thin; this is another expression of the imperfect development of the organ from the connective tissue standpoint. (See Figure 301.) It seems that the pulp, being housed in the pulp chamber, where there is no opportunity whatever under normal conditions for any touch of extraneous matter with its tissue, is poorly provided in the thickness of the walls of its blood vessels. This may also be due in part to the fact that the tissue is housed and completely fills the space of the pulp chamber, and the blood vessels derive some support in that way.

At any rate, the walls of the blood vessels are very thin and the muscular coating of the arteries is very slight. This being the case, any unusual blood pressure is liable to expand some of the arteries, while others collapse to make room, and also the veins may collapse in any such unusual blood pressure. This causes the pulp to be especially influenced by thermal changes. Each thermal change, whether it is to greater heat or to greater cold, produces a shock which calls to the pulp a greater amount of blood, causing a twinge of pain which soon passes away. This is normal to the pulp. This condition of the pulp and its arteries renders it particularly liable to hyperemia developed by excessive thermal changes, in which the pulp tissue becomes much overcrowded with blood. It is a condition that must be reckoned with in practice and will be treated more fully a little later.

NERVES AND NERVE FUNCTIONS.

The nerve supply is derived from the nerve filament, or filaments, which enter the apical foramen. These are also distributed principally along the layer of odontoblasts, and naked nerve filaments are frequently found among or between the odontoblasts, their endings seeming to be upon the pulpal ends, or among the cells of the odontoblastic layer. These nerve filaments do not, however, enter the dentinal tubules, so far as has yet been satisfactorily demonstrated. They give the pulp its sensitiveness, and the odontoblastic layer with its filaments conveys the sensitiveness from all parts of the dentin.

This is the one place in the minute anatomical structure of tissue in which sensitiveness without nerves seems to be demonstrable, and gives prominence to the idea that it is the cellular elements which are sensitive, and that it is the function of the nerve to convey sensitiveness. True, nerves may have sensitiveness of their own, but the conveyance of sensitiveness or other impulses is the important function of nerve tissue. The sensitiveness of the cellular elements is carried by them to the brain, and the expression of pain is produced upon the sensorium. That the cellular elements are themselves sensitive may be demonstrated by microscopic study of them under special conditions in which they can be seen while active.

The ameba displays this sensitiveness when it is in motion upon the stage of the microscope in a fluid particularly designed for this purpose. It will be seen to give out its pseudopodia and move about in the way of an ameba, picking up things which it may find in the water and taking them into its mass. While it is thus active a little jar upon the stage will cause the cell to suspend its work and roll up into a round ball. When allowed to remain quiet for a time, it will resume, and another jar will cause the same sensitiveness to appear.

As the ameba has sensitiveness within its own mass, so the cells in the higher animals, each has its own individual sensitiveness and responds to injury. It is the function of the nerve to convey this expression of sensitiveness to the sensorium, where it is registered as conscious pain, or conscious sensation. There is therefore a division between the function of the ordinary cell and the function of the nerve, though this does not prevent the nerve itself from having its own sensation; but it is a mistake to suppose that every sensation is a nerve pain. This will be understood better perhaps by the illustration, Figure 303. This

is an example of sensitive tissue in which no nerve is touched, as in sensitive dentin.

Sensory function of the pulp. In sensory function the pulp would seem to be similar to other connective tissues of the body. It exhibits pain upon touch, cutting or other injury of its substance. This pain is sharp and lancinating. The sensitiveness is more distressing than that of cutting the ordinary tissues; so much so that it is universally dreaded by patients. Every part of this tissue seems to possess this exquisite sensitiveness. I have had a number of instances in practice in which I could examine this critically in different parts of a widely exposed pulp. In different states of irritability wide differences are found in the sensitiveness of the pulp. I have examined a number of pulps when laid bare by accidents which have broken the teeth, exposing a considerable amount of pulp tissue. In some of these cases, seen early after the accident, the pulp was insensitive and blanched, showing its susceptibility to shock. The shock of breaking the tooth seemed to have obliterated sensation for the time, but by waiting for a time the sensitiveness returned and it became reddened, hypersensitive and painful. Then the slightest touch would give exquisite pain.

I think this history is repeated in almost every case of breaking a tooth and exposing much of the pulp in a sudden way. The pulp would not seem to be very much different from other tissues of the connective tissue group, and one would naturally suppose that the pulp would heal the same as other connective tissues. This point will be considered later.

In the ordinary diseases of the pulp its exquisite sensitiveness comes out boldly in the expressions of pain produced. Perhaps there is no bit of tissue in the body which becomes so sensitive as the pulp and exhibits more vague symptoms. This sensitiveness is perhaps due in large degree to the arrangement of its tissue, and the thin walls of its arteries and veins. In these the variations of pressure by the blood seems to have a marked influence, causing the pain to have a peculiar throbbing character much dreaded by persons who have experienced it.

The more sensitive nerve endings are along the line of the odontoblasts, and with the peculiarities of the tissue which have been expressed here, it will be seen that these are compressed against the walls of the hard dentin at every pulse-beat in these abnormal conditions. This seems to be an especially sensitive area of the pulp, and the compression of this layer of cells and of the fibrils which enter the tubules of the dentin produces

excessive pain when the pulp is much overfilled with blood. The pulsations of arterial pressure are pounding, as we may say, upon these sensitive points. This is to be reckoned with in the management of the diseases of the pulp.

Pain and touch.

We have seen that the pulp is completely enclosed in the central portion of the tooth — the pulp chamber and root canals — and entirely fills this space. It is surrounded by hard tissue in such a way that under normal conditions nothing extraneous can touch it, and would seem to be shielded very perfectly from outside influences, with the exception of changes of temperature. Under these conditions the pulp becomes an internal organ, and is subject to much the same conditions as to touch and pain as other internal organs.

The rule is that nature produces no functions which can not come into use. Consequently the sense of touch is absent from all of the internal organs, but resides in the skin and in the mucous membranes which line the entrances into the body, as the mouth, pharynx, etc., and the exits from the body. It exists in all parts that can ordinarily be touched by the fingers or by extraneous substances entering the body, but disappears as soon as those have fairly entered the body.

The sense of touch is a localizing sense and should be separated completely from the sense of pain. By the sense of touch we localize a touch on any part of the surface of the body. Pain in and of itself is not a localizing sense. Internal organs manifest pain in a vague way as to localization, and often the pain complained of is at some distance from the tissue in actual distress. Indeed we know nothing of our internal organs except what we obtain from education. A man has no idea of the existence of his stomach or of certain intestines from any natural sensations of his own, no matter how much he may have learned of these in an educational way, but in this way he learns to attribute certain sensations to certain organs vaguely, and is often wrong.

I remember the case of a friend who often complained of pain in the region of the gall bladder, and his physician had made a diagnosis of gall stones. A second physician made a similar diagnosis. In an attack of pain which was very much worse than the previous attacks, a surgeon was called. This surgeon spent quite a little time in examining the case, and said that an operation was an absolute necessity and that it should

be done at once. I noticed that when the patient was etherized and the surgeon took up his knife, he cut for the appendix, and he was right. This is one illustration of how one may be deceived, and how even well-informed physicians may be mistaken as to the location of internal injury or disease, by the manifestation of pain. Physicians and surgeons learn by their study of the peculiarities of pain, and particularly through their observation of the interferences of function, to make proper diagnoses in many cases in which the pain is not definitely located by the patient.

Pulp an internal organ. The pulp is an internal organ and shows all the vagaries as to its location of pain that are found in other internal organs. If a pulp has become exposed from decay in some hidden away locality, which has not been discovered by the patient or dentist, the patient is liable to locate that pain anywhere on that side of the face. It may be in the teeth or in the jaws, or at some distance from the teeth in the face, head, ear and various other localities, and may display a peculiar disposition to appear first in one place and then in another. This is liable to lead to errors in diagnosis unless it is understood and carefully guarded.

It is common for patients to refer pain to another tooth than the one which is diseased, or even to the opposite jaw. If the diseased tooth is in the lower jaw, the patient may refer the pain to a tooth in the upper jaw, on the same side of the mouth. This is comparatively common in cases where the diseased tooth has a cavity in a position unsuspected by the patient, as in the proximal surfaces near to or partly covered by the gingivæ. If there is another tooth in the same side of the mouth known to have a cavity, the patient is very liable to attribute the pain to this other tooth, an examination of which may show that the cavity is not of sufficient depth to be liable to produce inflammation of the pulp.

Once during my practice a good Irish lady applied to me. She had been suffering very severely for some weeks with violent toothache which she attributed to one of the bicuspids in the upper jaw. In listening to her, I found that she had already been to two dentists, both of whom had refused to extract a tooth for her; or rather, she would not permit them to do so, because of a disagreement as to the tooth causing the pain. The dilemma thus plainly presented to me, and the fact that the poor woman was almost crazy with pain, induced me to extract the first molar in the lower jaw instead of the upper bicuspid, without saying a

word to her about it. She was very wroth, and gave me several pretty sharp pieces of her mind. I finally told her to come and see me three days later when we could talk the matter over. I knew full well that her pain would cease, and she came back and acknowledged that it had ceased, but how it was that extracting the tooth in the lower jaw stopped the pain in the upper jaw, she could not understand "a tall, a tall."

Pain of other diseases may simulate pulp pain. There are other diseases with which pulp pain may become confused because of the similarity of the symptoms. In cases of *tic douloureux,* a true neuralgia of the fifth pair of nerves, particularly of the second and third branches from which the teeth are supplied, the pain may be confused with that caused by inflammation of the pulp. This word *tic* means a twitch, and *douloureux* means pain. The twitching is pure pain — nothing else that can be discovered. It is paroxysmal and the paroxysms are very prone to be brought on by any slight touch on the face, or touch of the hairs of the mustache, or a breath of cold air. Cases in which the symptomatology is confusing should be referred to a specialist. A full discussion of this condition would be out of place in this book. One in ordinary practice sees but few cases of tic douloureux; indeed, one may spend a lifetime in the practice of dentistry without seeing a case, but he is liable to be confronted with a case at any time, in which he will have to make a diagnosis between inflammation of the pulp and this painful twitch.

Patients will give descriptions of pain which they refer to the teeth, but which they also refer to other parts. These descriptions are often given in language so vague that it is very difficult to follow them.

I once had a patient who complained of a twitching sensation in his stomach, or in the region of the stomach, and his physician had been treating him for this without effect. Finally there were some symptoms about the head and jaws, and the physician brought the patient to me for an examination. In a very thorough examination, I found a point where there seemed to be no support to the soft tissue over a portion of the root of a tooth, and on pushing a sharp instrument through this it dropped into a cavity in the root, and a scream from the patient indicated that I had touched something unusually sensitive. I immediately extracted the tooth and found a large absorption cavity exposing the tissue of the pulp. Indeed the tissue of the pulp itself was being absorbed. This ended the man's trouble.

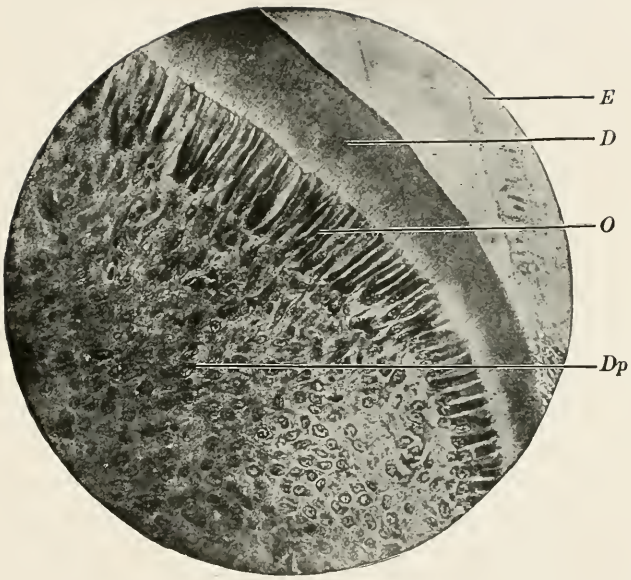

FIG. 299.

FIG. 299. Odontoblasts and forming dentin: E, Forming enamel. D, Forming dentin. O, Odontoblasts. DP, Body of dental papilla. (From photomicrograph by Rose.)

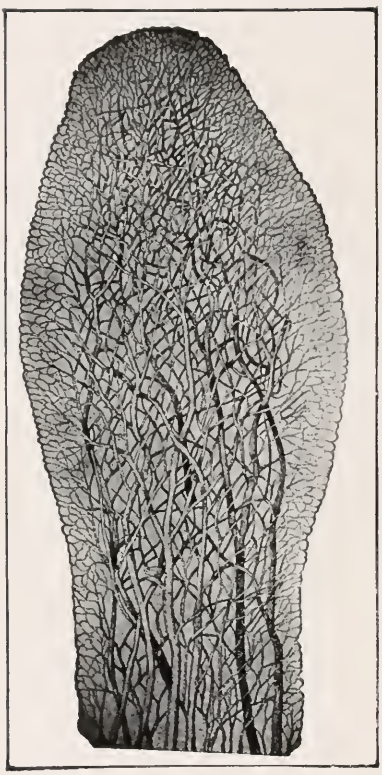

Fig. 300.

Fig. 300. Diagram of the blood vessels of the pulp. *C. H. Stowell.*

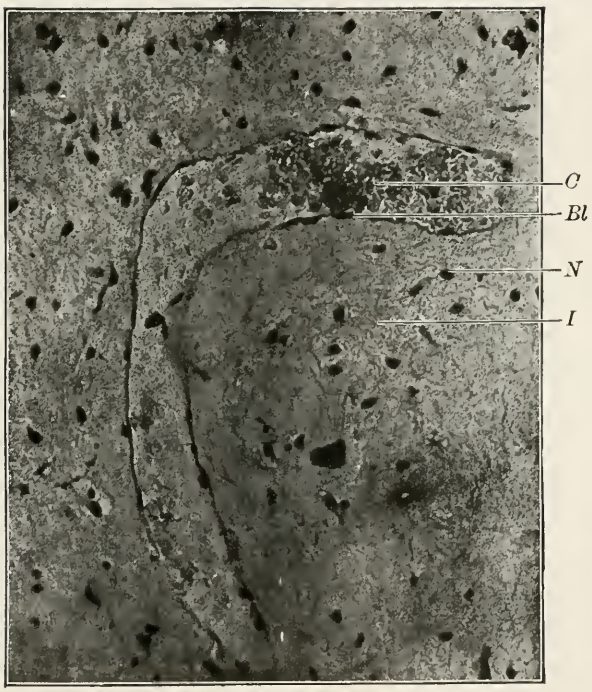

Fig. 301.

Fig. 301. A pulp blood vessel, showing the thin wall: c, Blood corpuscles in the vessel. Bl, Blood vessel wall showing nuclei of endothelial cells. N, Nuclei of connective tissue cells in the body of the pulp. I, Intercellular substance, showing a few fibers. *Noyes.*

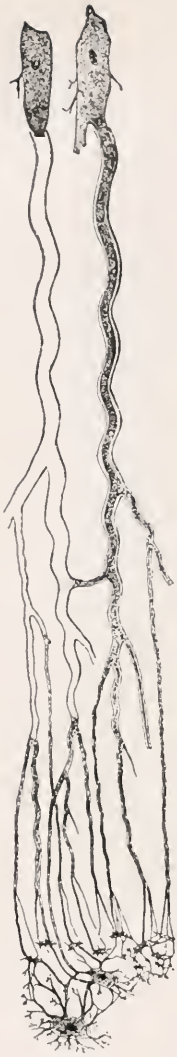

Fig. 302.

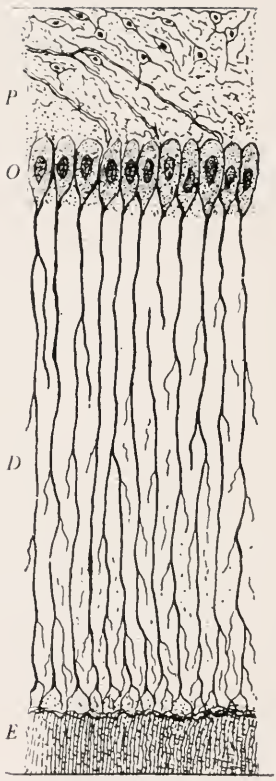

Fig. 303.

Fig. 302. Diagram of odontoblasts and dentinal fibrils. *C. H. Stowell.*

Fig. 303. A diagram illustrating sensation without nerves in the dentin. E, Enamel. D, Dentin. O, Layer of odontoblasts. P, Pulp of tooth with nerve endings in physiological connection with the odontoblasts. The fibrils of the dentin are prolongations of the odontoblasts. Any injury to them is an injury to a portion of the odontoblasts and is transmitted by the nerves to the brain.

These illustrations are given simply to point out the vagueness which occurs in internal organs as to expression and localization of pain, and the tooth pulp must be considered an internal organ.

Sense of touch is in peridental membrane. Touch is a localizing sense. Its function is to point out closely the part of the body touched and this is done more definitely in some regions than in others. If the surface of a tooth is touched the sensation is felt. If the pulp is removed and the tooth is again touched, there is no difference in the sensation. The effect of touch upon the tooth is not destroyed or changed by removing the pulp because the sense of touch for the tooth is within the peridental membrane. If the peridental membrane is diseased, a touch upon the tooth may be felt both as touch and pain, because of the inflammation. The response is registered on the sensorium as both touch and pain. All of this should be kept in mind in making examinations. Many of these things will be considered in discussing the diseases of the pulp.

Healing powers.

The description I have given thus far of the tissues of the pulp and of its functions would indicate that the power of healing is very low in this organ, and this agrees with clinical observation. Indeed, the rule is that in the adult tooth any touch of the pulp tissue in excavating cavities which is sufficient to draw blood, will eventually destroy the whole of the pulp. Such an injury fails to heal by any treatment yet devised. Some exceptions to this rule may have occurred under peculiarly favorable conditions, but the rule holds good in such a large majority of cases that we must regard these as exceptions. This is partly due to the fact that the mutilation in these instances is of the very sensitive odontoblastic layer, the regeneration of which when injured, is in great doubt. It is possible that in the child's tooth, before the full development of the roots, some of these injuries may be bridged over and in a very short time become perfect again, not by the multiplication of the odontoblasts, but by the falling together of the odontoblastic layer of the neighborhood and straightening into normal form. During this formative period the pulp chamber is becoming narrower and narrower, and the odontoblastic layer is becoming more crowded in the whole extent of its surface, making a more favorable opportunity for this bridging-over process in the growth of the dentin. In the adult tooth there is practically nothing of

this kind to favor the healing process, and it is rare that wounds heal. They generally produce an inflammatory movement which in the end destroys the pulp.

History of efforts to save exposed pulps by capping. I have seen cases in which widely exposed pulps have been capped and the results should be regarded as successful, although they could not be taken as a guide for treatment. I remember once finding, in the mouth of a very intelligent person, an amalgam filling in a lower molar that was then in such a state of apical pericementitis that in my judgment an alveolar abscess could not be prevented. The patient told me that the pulp of this tooth had become widely exposed fifteen years before, and that Dr. C. W. Spalding, then in practice in St. Louis, had capped it with a piece of gold plate and made a filling against this capping, and the tooth had done service, and had been comfortable, during the intervening time. I made a cut across the filling and broke it out of the cavity. I was surprised to find that the inflamed pulp had evidently protruded into the portion of the cavity left vacant under the piece of gold plate, filling it completely, and all of this portion of the pulp had become calcified. The calcification of the pulp had continued until the pulp tissue was strangled (I think that is the best term to use) by the amount of secondary dentin which had been formed. The pulp dies in this way in cases of extensive abrasion, as a result of which the pulp chamber becomes filled with calcific material. The same result follows extensive erosion, or sometimes occurs without either abrasion or erosion, and finally leads to the death of the pulp.

This was a case of what might be termed the most radical operation of capping a widely exposed pulp, which nominally would be considered a successful case. However, the percentage of such successes would be so small that no practitioner would be justified in pursuing such a course. The accumulation of records of such procedures seems to be sufficient to condemn such efforts.

Although but few dentists would to-day recommend the capping of pulps in adult teeth, there have been several attempts to revive the practice, generally by those unfamiliar with the history of these cases, and for this reason I will add a report of my own observations made many years ago.

Dr. Witzel, of Germany, wrote a considerable volume upon

this subject,* in which he advocated various means of treatment for exposed, inflamed and suppurating pulps, and even the extreme procedure of the removal of the bulb of the pulp found in a state of suppuration, retaining the root portions of the pulp and capping them over. Other writings as extraordinary as this may be found scattered through our literature. Happily they have had very small influence in inducing men to accept and practice such procedures, and I think it is now generally understood by the dental profession that such treatments are pernicious.

My observation of this has been long and continuous. Quite early in my practice it became my habit to have my assistant place on my desk the record of each patient for whom I had previously operated, so that I could see at a glance what I had done. This led me to question understandingly all cases in which I had capped pulps, and to make a note of the date of examination. During the years previous to 1870 I had made a good many of these pulp cappings and had records of the results. These records showed the loss of the pulp from death in all cases in which I had made cappings for adults.

These cappings were made by the process most commonly used by dentists at that time, excepting that perhaps I was somewhat more careful to remove all decayed dentin than many others. The pulp tissue was exposed by removing the softened dentin from over it, or from around it, if it was fully exposed to the access of the saliva, which most of them were not, for I did not often use a capping in such cases. This was done with a very sharp, broad-bladed spoon excavator, which would not force the debris into the pulp tissue. Then the cavity was flooded with phenol, and when wiped out I was careful to note whether or not the pulp tissue was whitened. If it was not, another application was made after carefully drying the cavity, for sometimes it was found that there was an exudate from the pulp which would prevent contact with phenol. Then a capping of chloride of zinc was placed. This material was then sold under the name *Os Artificial.* We did not at that time have the phosphate cements. The chloride of zinc would cause considerable pain unless the pulp had been protected by the eschar produced by the phenol. This material was always made soft enough so that it could be flowed into the cavity and produce

* Compendium der Pathologie und Therapie der Pulpakrankheiten des Zahnes, von Dr. Med. Adolph Witzel, 1886.

little or no pressure, differing in that respect from the phosphate cements being used to-day.

I give the results in my cases at that time, published in the Transactions of the Illinois State Dental Society for 1870, page 6, as follows:

"Of all the cases treated in 1860, only forty-two have remained under my care, so that I can trace them with certainty. With these the result has been as follows: Seven abscessed within one year, nine were found with pulps dead or abscessed in the second year, eleven in the third year, seven in the fourth year, two in the fifth year, one in the sixth year, one in the eighth year, and four of the pulps remain alive to-day.

"In counting these, I have only taken those which were at the time considered successful cases. Those that failed within one month are not included. The immediate failures at that time will outnumber those of to-day, on account of the greater imperfections in operating.

"It is very important to notice the ages of these patients, and the varying rate and time of failure. In eighteen cases they were under twenty years of age, while in twenty-four cases, they were over twenty years of age. Of those under twenty, six failed* in the first year, four in the second year, two in the third year, one in the fourth year, and four are successful after ten years. Of those over twenty, one failed in the first year, five in the second, nine in the third, six in the fourth, two in the fifth, one in the sixth, and one in the eighth year.

"It will be seen that all the cases in patients over twenty years of age have failed, and that in the successful cases, the patients were under that age.

"In the successful cases the fillings which were placed at the time of the capping, were removed, two in the second year, and one in the third year, and the teeth refilled, when the pulps were found covered with solid dentin which had been thrown out over them. In the eighth year, the fourth successful case was found in like condition.

"The cases reported as failures did not all suppurate; but in some of them the old fillings had to be replaced by the new, and the pulps were found dead. My record, unfortunately, only shows which teeth had the roots filled, or were extracted, without any reference to their suppuration, so that I can not state

* The word "failed" in this report represents the time at which these failures were discovered.

the exact number that did actually suppurate." My memory is that most of these cases were found with alveolar abscess.

Many dentists of my acquaintance were using this particular mode of capping pulps. A very large proportion of the pulps thus capped gave no symptom of inflammation. Very few practitioners of that time kept records of such cases, and when patients returned to them they were often unable to tell which teeth had had pulp cappings, and which had not; they were even unable often to determine whether they had made certain of the fillings. They were therefore unable to know the results of their operations.

At that time the discussions of the Society were not taken in shorthand, and do not appear in the printed transactions, but I do know that in closing the discussion which followed this paper, I urged the members of the Society to make records of such cappings, also of the conditions observed when patients returned for examinations, in order to learn definitely what successes were attained.

This process of capping was very generally abandoned within a few years because so many pulps died, and the general practice swung to the opposite extreme, of removing every exposed pulp.

In the report given above, all of the pulps which lived were for persons under twenty years of age, and all but one were for persons under fourteen years. Therefore, the successful cappings were those of the pulps in children's teeth, the root canals of which were still large. In more recent years, I have confined the operation of capping to those cases in which the exposures have occurred very early in life, because they offered the greatest probability of success. The technic of capping pulps will be given under the discussion of pulp treatment.

DISEASES OF THE DENTAL PULP

ILLUSTRATIONS: FIGURES 304-341.

THE most common diseases to which the dental pulp is liable are hyperemia and inflammation. To these may be added hypertrophy, which occurs in some cases of chronic inflammation; traumatisms, including blows upon the teeth, broken teeth, and injuries to the pulp in the excavation of cavities caused by decay; and the calcifications which occur, either growing upon the walls of the pulp chamber, or within the pulp tissue unattached to the walls. These calcifications may cut off the pulpal ends of the dentinal fibrils and destroy the life of large areas of dentin, perhaps in some instances including the entire crown portion of the tooth. These occur most as a result of abrasions or erosions of the teeth, but may occur without them. This practically covers diseases of the pulp observed by dentists in practice.

Historical Statement.

Previous to the publication of the American System of Dentistry in 1886, there seems to have been much speculative consideration of inflammation of the pulp, but a review of the literature reveals the fact that practically no studies of hyperemia of the pulp had been made before that time. Therefore, most of the cases of hyperemia must have been classed as inflammations. It does not appear that the diagnoses were verified by careful microscopic examinations of the prepared tissue, combined with previous studies of the symptomatology. If they had been so studied, we can not conceive that so many errors could have occurred. Indeed it would seem that most men have considered that when the pulp was painful, it was necessarily inflamed.

This is far from correct, for many pulps which are painful, are painful from hyperemia. Many pulps which are inflamed are painful from the hyperemia accompanying the inflammation, and the symptomatology is very similar to that of hyperemic pulps. If one should extract a painful tooth and afterward break it open and examine the pulp with the naked eye, or hand

lens, and find it very red or turgid with blood, and on such an examination pronounce it inflamed, most of the pulps from aching teeth would be classed as inflamed. Hyperemic pulps will show the same turgescence. For this reason, the mistakes in classifying hyperemic pulps as inflamed pulps seem to have been common in the past.

A number of writers have given extended classifications of inflammations of the pulp, apparently based on microscopical examinations of the tissue. These can not be distinguished clinically, and, therefore, are not useful to the general practitioner, so far as his every-day operations are concerned. They are interesting as scientific studies, and in the course of time a more critical observation of the symptomatology, as compared with the particular form of the inflammatory process, as studied in prepared sections, may become of more importance than at present.

From the standpoint of practical treatment, it is important that a different diagnosis be made, whenever this is possible, between simple hyperemia and inflammation, because the effort should be made to save all hyperemic pulps, while practically all inflamed pulps must be destroyed and removed. Hypertrophy of the pulp due to chronic inflammation, in which the pulp tissue is protruded through the orifice into the cavity produced by caries until it fills, or partially fills, the cavity of decay, is always easily diagnosed. Those cases due to traumatism are usually recognized without difficulty, and the character of the traumatism will indicate the treatment.

In the consideration of the hard formations, it is of most importance that certain conditions which are known to cause these calcifications be recognized early and treatment promptly applied. A better knowledge of the various forms of these hard formations will also be of assistance in facilitating their removal.

To show the extent to which many have gone in describing inflammation of the pulp and the varieties assumed by it, I copy here from "Zahnheilkunde," by G. Preiswerk, published in 1903, the following from an index given in the first pages of the book, "Die Erkrankungen der Pulpa":

		PAGE
1.	Hyperaemia pulpae	268
2.	Pulpitis acuta superficialis	271
3.	4. u 6. Pulpitis acuta partialis, totalis und traumatica	271

5. u 8. Pulpitis acuta partialis purulenta und Pulpitis chronica totalis purulenta..... 274
7. Pulpitis chronica parenchymatosa..... 275
9. u 10. Pulpitis chronica hypertrophica granulomatosa und sarcomatosa..... 277
11. Gangraena pulpae 278
12. Pulpitis idiopathica seu concrementalis..... 280

While most of the conditions mentioned can be made out in an examination of pulps after obtaining and making sections of the tissue, I have failed to make them out by diagnostic signs before removing the teeth and bringing the prepared pulp tissue under the microscope.

I have taken careful notes of the symptoms in cases in which there was hyperemia or inflammation of the pulp and from these have written my conclusions as to the condition of the pulp. Then I have extracted the teeth, prepared the tissue and brought it under the microscope for study. I have repeated this process for a great many teeth, both before and since my writing in the American System of Dentistry, and my conclusion, after all of these studies, is that inflammations of the pulp in earlier stages can not be differentiated from hyperemia; furthermore that it is impossible to determine from the symptomatology, whether or not inflammation exists in the pulp. Many pulps which are inflamed and suppurating give no symptoms whatever. In my examinations I classed widely inflamed pulps as hyperemic pulps, pure and simple. Indeed, so many errors of diagnosis occurred that I am satisfied that we are unable to differentiate between special conditions of inflammation, as it occurs in the pulp, by symptomatology. The only reliable method of differentiating between hyperemia and inflammation of the pulp, as will be explained later, is by determining whether or not the pulp has been actually exposed.

Therefore, such classifications of the inflammatory conditions of the pulp as the one quoted can be of no benefit whatever to the dentist in his every-day practice. In this book, I shall adhere to very much simpler divisions of the process of inflammation, as it occurs in the pulp. I shall make a few changes in classification from what I wrote for the American System of Dentistry in 1886. One of the most prominent of these will be the classification of every pulp exposed to carious dentin, as an inflamed pulp, whereas in that writing I classified only those exposed to the saliva as being always inflamed pulps.

Personal Studies of Hyperemia and Inflammation of the Pulp.

In the American System of Dentistry, I wrote the chapter on the Pathology of the Dental Pulp,* and on reviewing my presentation of the subject, I find that I will have but little new or different to present now.

In the studies at that time, I made microscopic sections of a great many hyperemic and inflamed pulps, including those in which the disease had become severe. In all of the cases there was a determined effort to capture the conditions of the blood which was present within the pulp with its expansion of arteries, or without this expansion, and keep careful notes of the symptomatology in each individual case at the moment the tooth was extracted, also of the occurrence of paroxysms of pain, and their severity. This record was kept of each such case from the time the patient first presented until the tooth was extracted.

In some cases, the greatest degree of pain which I could produce by the application of heat or of cold, was brought about, and the tooth extracted at the moment the pain seemed greatest. Immediately after the extraction, the tooth was caught with one finger upon the occlusal or incisal edge, and one on the apex of the root, and held so for a few moments to prevent more blood from issuing from the pulp. It was then dropped into Miller's fluid, or some such fixative. Observation showed that, when so treated, no more blood would be lost from the pulp. A label was attached to the tooth by a string which hung out of the jar, so that I could put a number of teeth into one large jar of the fluid.

In other cases the teeth were extracted when there was no pain, in the interval of quiet between paroxysms, and were treated in the same way. I collected specimens of teeth that were showing moderate hyperemia, both in the stage of excessive pain and in the stage of quiet, the various cases representing practically all the features of the pain in hyperemia, including pulps that were on the point of being destroyed by the disease. I also included a number of cases in which the pulp had just died, or had very recently died, from this cause.

Technic of preparing specimens.

After a sufficient time in the Miller's fluid for fixation and considerable stiffening of the tisssue, the teeth were cracked in a strong vise, placing them so as to produce pressure on the

* American System of Dentistry, Vol. I, p. 829.

greatest portion of two sides, usually the lingual and labial of the front teeth, which were most generally used for this purpose.

After cracking the tooth in the vise, it should be placed under running water and washed moderately free from the Miller's fluid in which it has been soaked. Sometimes, with the best of care, the pulp tissue will be crushed and ruined, but in the majority of cases, as I have handled them, the tooth will be split lengthwise, exposing the larger portion of the pulp in one or the other half. Sometimes a part of the pulp will be held in one half, and another part in the other half. A fine needle may be used to advantage to free the pulp tissue from one of the halves, without injuring it. Then with the pulp lying in a single half of the broken tooth, one may tease it out slowly and carefully, trying always to pass the needle between the pulp and the walls of the pulp chamber so as not to disturb the relations of its tissue.

In this it will be discovered, if one is using a magnifying glass and working very carefully, that the fibrils are being pulled out of the dentin in some positions. Over other portions of the pulp odontoblasts will be pulled off from the pulp and remain adhering to the walls of the pulp chamber. It is quite desirable to remove as much of the odontoblastic layer with the pulp as possible. When this has been done, the pulp is placed in the washing tray and allowed to remain in running water until all traces of the discoloration by the Miller's fluid have been washed out. The bit of tissue is then ready for the usual preparation for sectioning and staining, which does not differ from that generally used in the preparation of other tissues. It is passed through the usual alcohol solutions, beginning with 45 per cent, then 60 per cent, 80 per cent, 95 per cent and finally remaining some time in absolute alcohol. Then it may be blocked in paraffin or celloidin.

I have also cut much of this very delicate material in gum arabic. A solution of gum arabic is made and filtered. A sufficient portion of this is placed in a dish and the pulp laid in. The amount of gum arabic in the solution should always be such that the pulp tissue will sink. It should never float. If it floats the gum arabic solution should be diluted with water and given time for this to become equally distributed before finally placing the specimen in it. Then the gum arabic solution should be placed where there will be a very slow evaporation of the water. It will become thicker and thicker from the evaporation. It should require several days in order that the pulp tissue may be

thoroughly filled with gum arabic. The gum arabic is very prone to decomposition and to growths of mold, which may be prevented by a few small crumbs of camphor distributed over the surface. When the solution has become sufficiently stiff that it may be built up on a cork, or other suitable block which may be held by the jaws of the sectioning machine, this should be done, and the pulp carefully lifted and placed upon this as it dries a little. Then, as this material is clear, and the position of the pulp may be accurately seen, it should be covered with more of the thick solution, so that the pulp will be well buried in it, and then it should be manipulated so as to bring the pulp in the exact position wanted for sectioning.

In this condition it is floated by attachment to a cork, if the cork has not been used to begin with, on the surface of alcohol. The alcohol will take the water from the gum arabic and cause it to become hard. A solution of about 70 per cent alcohol should first be used. Within a few days it may be increased to 95 per cent. Careful examinations of the mass should be kept up during this time, to see to it that it is not rendered too hard and brittle. When it is judged to be hardened sufficiently, the whole should be removed from the alcohol and covered very closely to prevent further drying of the surface. It should remain thus covered for a day or two, because the surface of the gum arabic will at first be dried very much more than the interior, and during this time of waiting, the drying will be equalized so that the whole of the specimen is of equal hardness. It requires some experience with this material to attain just the exact conditions which will give the best results.

When the sections are cut, laid on a cover-glass and moistened with water, they quickly unfold, and will usually lie in very perfect position. The gum arabic dissolves in the water, and this may be drained later, leaving the section upon the cover-glass without disturbance. It is well to have the cover-glass covered with some substance that will cause the section to cohere, as equal parts of egg albumen and glycerin. Then the staining can be carried out the same as in staining other sections of tissues, and brought under the microscope in good condition for examination.

This is a brief detail of the plans I have used in studying the conditions of the pulp in hyperemia, inflammation, in suppuration, and in fact all of the diseases known to it, and these studies are the basis of most of the descriptions of the tissues and tissue changes in diseases of the pulp, which I shall give.

HYPEREMIA OF THE DENTAL PULP

ILLUSTRATIONS: FIGURES 304-307.

Hyperemia of the pulp consists essentially in the expansion of the blood vessels, principally the arteries, during any sudden abnormal blood pressure. This is of frequent occurrence; so frequent, indeed, that cases are almost constantly presenting. In general, hyperemia is seen as an accompaniment of another disease, and is not regarded as a disease in itself. If, however, a femoral artery is tied on account of an aneurism, and the circulation in the leg proves insufficient because of this, a general passive hyperemia of the leg may occur, often with fatal results. The venous hyperemia, which gradually approaches stasis, becomes a pathological condition, rather than a symptom. The hyperemia of the tooth pulp, in distinction from this, is always an arterial hyperemia.

Etiology.

The most frequent cause of hyperemia of the pulp is a sudden change of temperature. Any sudden change from the normal temperature, either too hot or too cold, seems to affect prominently the blood pressure in the pulp, causing an inrush of blood which gives a more or less sharp twinge of pain for the moment, and then passes away. This is a *physiological hyperemia* of the pulp. Practically every normal pulp will give this expression when either ice-water or a hot drink, or very hot or very cold food of any kind, is taken into the mouth. This is not abnormal, but normal to the pulp. *Pathological hyperemia* is an extraordinary excitation, in which the function is forced beyond normal limits, and the larger majority of cases are brought on by sudden exposures to heat or to cold, which are sufficient to cause this normal function to become excessive and morbid.

There are some other conditions which produce hyperemia of the pulp. Irritation of the dentinal fibrils in the beginnings of caries seems sometimes to render the pulp more excitable, and may serve to precipitate a case of hyperemia. This occurs most frequently as a result of broad gingival third decays in

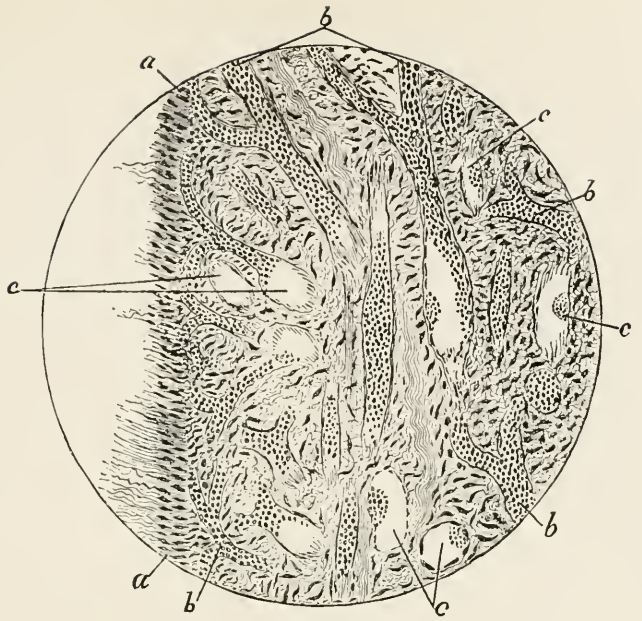

Fig. 304.

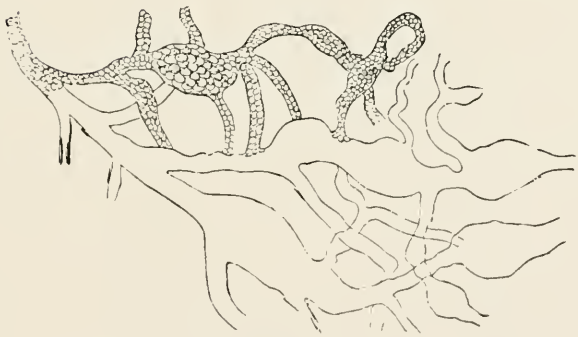

Fig. 305.

Fig. 306.

Fig. 304. Hyperemia of the dental pulp, showing the natural injection of the vessels: a, a, Layer of odontoblasts. b, b, b, b, Vessels distended with blood. c, c, c, c, Points from which the blood has fallen in handling the section.

Fig. 305. Dilated blood vessels from the dental pulp in hyperemia, from tooth extracted during a paroxysm of intense pain.

Fig. 306. A small vein from a hyperemic pulp, greatly distended and nodulated.

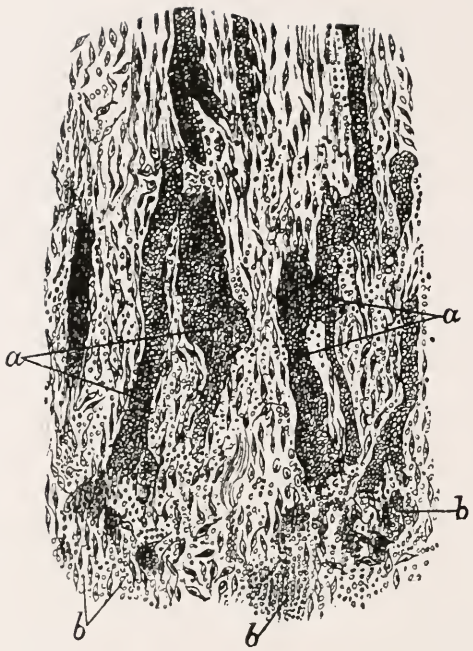

Fig 307.

Fig. 307. Section of hyperemic pulp, showing aneurismal dilatations of the vessels, extravasations of blood, and red blood disks escaped apparently by diapedesis: a, a, Dilated vessels. b, b, b, Extravasated blood. Besides this, red blood disks are plentifully distributed everywhere in the neighborhood of the veins. The tooth was extracted during a paroxysm of pain.

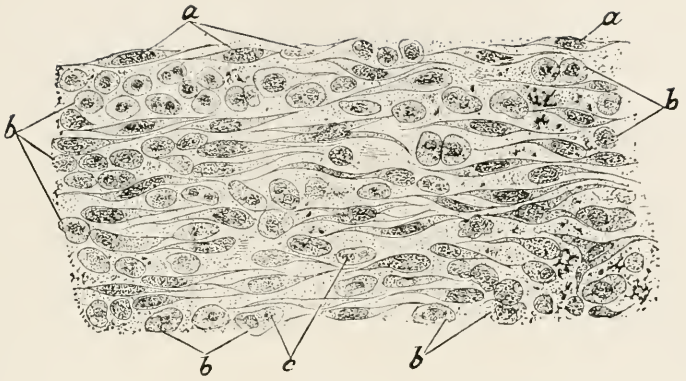

Fig. 308.

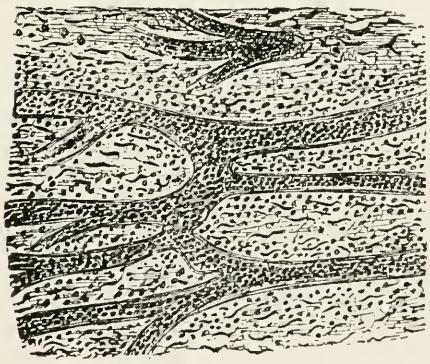

Fig. 309.

Fig. 308. Inflammation of dental pulp: a, a, Normal cells. b, b, b, b, Inflammatory elements. c, Cells in process of division.

Fig. 309. Section of dental pulp, showing dilation and congestion of blood vessels and escaped corpuscles.

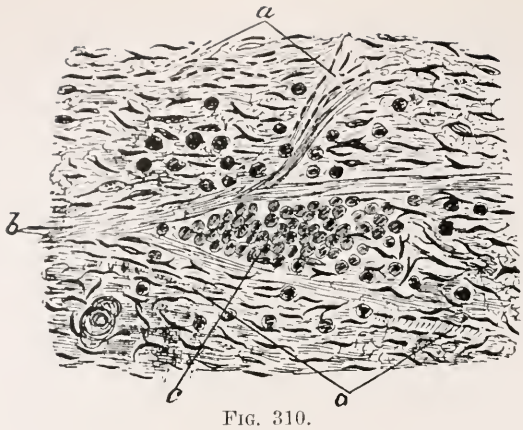

FIG. 310.

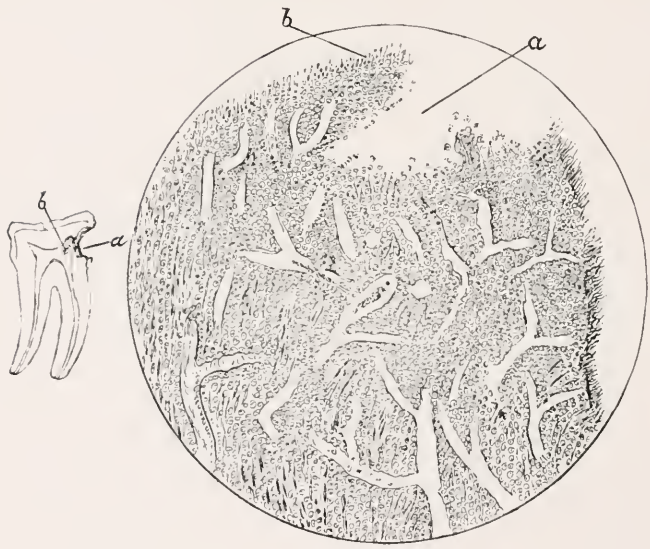

FIG. 311.

FIG. 310. Minute inflammatory focus within the tissues of the pulp: a, a, Arterial twigs. b, A nerve bundle. c, Collection of leucocytes.

FIG. 311. Diagram of lower molar, with caries at *a* which exposed the pulp. The darkened portion at b shows the extent of the inflammation. The rest of the organ was free from inflammatory change. Within the circle the inflamed tissue of the pulp is shown, a part having been destroyed by suppuration at *a*. The odontoblasts are undermined at b. The blood vessels which were filled with blood clot in the section are left blank here, that they may be more apparent.

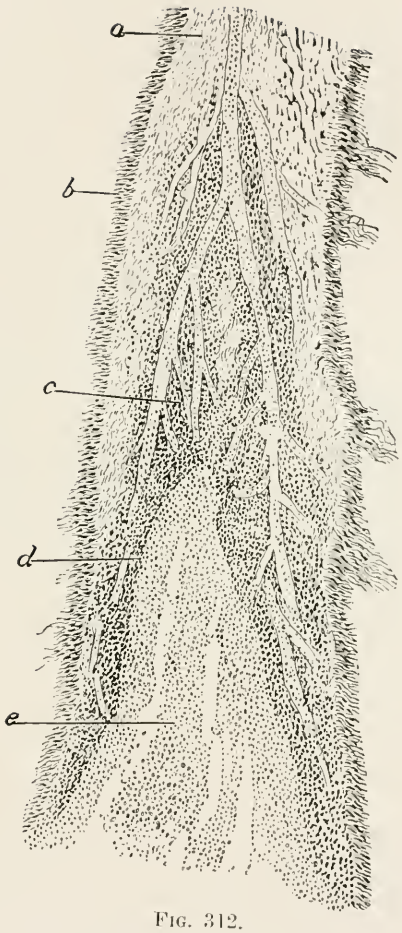

Fig. 312.

Fig. 312. Progressive suppuration of the pulp of an incisor: a, Healthy tissue. b, Odontoblast layer. c, Inflamed tissue in which the veins are seen to be dilated. d, Line of demarcation of the suppurative process. e, Pus. A part of the crown portion of the pulp had been destroyed by suppuration, and in the remaining portion it will be noted how the pulp is hollowed out, the process pursuing the course of the veins and converging to the center.

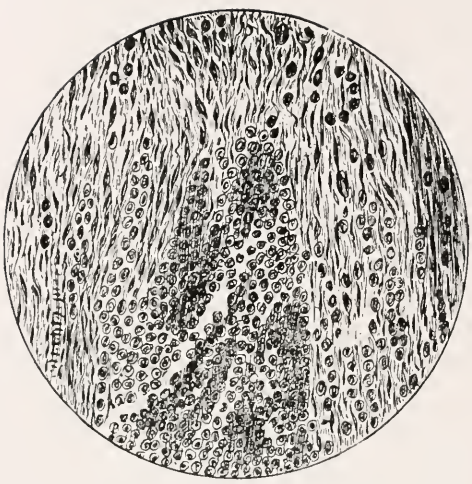

Fig. 313.

Fig. 313. Abscess within the tissues of the pulp. The field includes about one-half of the little pocket of pus.

Fig. 314.

Fig. 314. Chronic inflammation of the pulp, arcolation and degeneration.

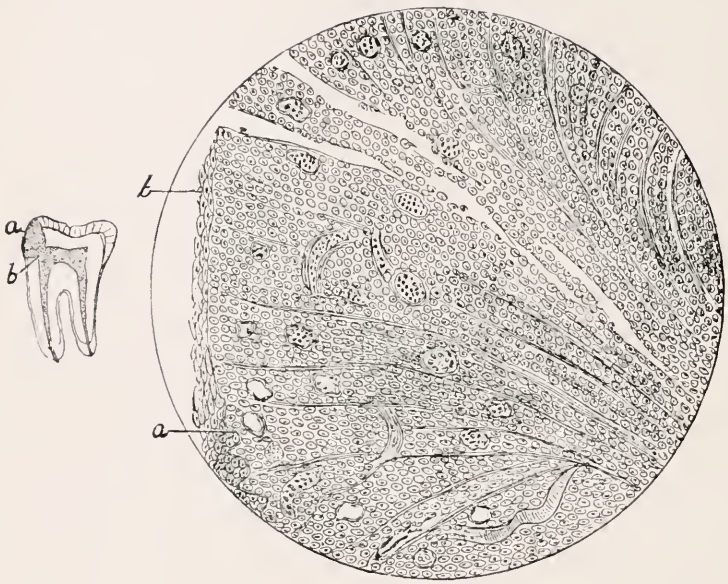

FIG. 315.

FIG. 315. Diagram of a lower first molar, with a cavity at *a* completely filled by a hypertrophy of the pulp, which has grown out through the orifice, exposing the pulp at b. In the circle, the hypertrophied pulp tissue is shown. It is composed almost entirely of granulation tissue of a very primitive type: a, a, Covering of epithelium presenting papillæ. b, Epithelium apparently without papillæ.

the buccal surfaces of the bicuspids or molars, or in the labial surfaces of the incisors, in which the ends of many dentinal fibrils are laid bare.

Hyperemia is very commonly the result of heat generated in polishing fillings, either by rapidly revolving disks, or by the vigorous drawing of tapes back and forth on proximal surfaces. A filling which is placed over a nearly exposed pulp may cause hyperemia by the rapid conduction of thermal changes. A very large filling may cause a hyperemia on account of the broad contact of its inner surface with many dentinal tubules, even though the changes of temperature in the mouth are not extreme. The grinding away of the enamel of a vital tooth will often cause hyperemia, either on account of the heat or by the irritation of the ends of the dentinal fibrils at the dento-enamel junction, even though the tooth is kept cool by a jet of water or otherwise. In the so-called oral prophylaxis treatments of the more radical nature advocated by some, in which portions of the enamel, whether decayed or not, are ground away to make it smooth, a very definite hyperemia will result in many cases, and this should always be a warning that the vitality of the pulp is endangered.

I have sometimes created hyperemia in testing teeth to determine the vitality of the pulps, by applying gutta-percha which was too hot. This produced severe pain at the moment, and the teeth were hypersensitive for some time afterward.

Pathological changes.

In the majority of the milder cases of hyperemia in which the teeth have been extracted during the time of freedom from pain, practically nothing abnormal is found. They would be passed as normal in any collection of microscopic specimens of tissues of the pulp; but in similar cases, in which the teeth were extracted during paroxysms of pain, considerable blood over the normal amount is always found in the arteries, and some of the arteries are more or less expanded in parts of their course. (See Figures 304, 305, 306 and 307.)

Some cases, in which the history showed the paroxysms of pain to have been longer in their duration, and in which the paroxysms themselves had been very severe, have shown more blood than normal, and expansion of arteries, even though the teeth were extracted during the time of the most complete cessation of pain. In those cases in which the tooth was extracted at the moment of the severest pain, the normal arrangement of the arterial system of the pulp was almost completely destroyed.

Some arteries were greatly expanded and filled with blood, while others near by were collapsed. Distortions of this character were seen throughout the tissue of the pulp. In none of these was there any actual sign of inflammation.

In those cases in which the pulp had just died, or in which its death had occurred within a few hours previously, this distortion of the tissues of the pulp was very much greater, and in addition to this the tissue was generally very much filled with red blood corpuscles which had passed through the walls of the blood vessels into the tissues, causing the pulp to appear almost like a blood clot.

Symptoms.

The symptoms of hyperemia consist of varying degrees of pain. Many cases produce a moderate excess of pain in a particular tooth, or in several teeth, which endures a little longer and is more severe than would be called the normal condition of pain in taking food or drinks that are hot or cold. Some patients are much disturbed when a tooth is sensitive to heat or cold, even though the increased sensitiveness may be of short duration. From this slight degree of hyperemia there may occur paroxysms of pain which will last for some minutes, or even half an hour or more, and then subside. Occasional cases occur in which there is more or less pain almost continually, and changes of temperature of three or four degrees are sufficient to cause paroxysms of extreme pain.

All cases of pain which may be induced by hot or cold applied to the tooth should be considered as hyperemia, provided there is no exposure of the pulp to carious dentin. If decay has reached the pulp, it should be considered as an inflamed pulp.

I recall a case in which a patient presented with an acute abscess, and she remembered that the tooth had been extremely sensitive to thermal changes for a few weeks after it was filled. She also stated that the tooth had since been perfectly comfortable until the day or two before the abscess developed. My record showed that I had placed the filling in this tooth eighteen years previously, and there is little doubt but that the pulp died very soon afterward. It had remained during the intervening years without involvement of the periapical tissues.

Sequelæ.

As the paroxysms of pain become more severe or of longer duration, some of the arteries are generally expanded, while

other arteries and the veins are collapsed to make room. It is but one more step to a complete stagnation of the circulation in the pulp, and the death of the organ. This is called *infarction.*

When a pulp dies of hyperemia, there occurs a solution of the red corpuscles which have escaped into the tissues. This coloring matter penetrates into the dentin, often causing a marked discoloration of the tooth, which is very difficult to bleach. This discoloration may be compared to the discoloration of the soft tissue which occurs about a contused wound. In the latter, however, the color is gradually changed to normal by the circulation, but a similar change can not occur in the tooth. When the death can be immediately discovered and the pulp removed, the discoloration may be prevented.

Cases which terminate in the death of the pulp often result in the formation of either an acute or chronic alveolar abscess. When the pulp dies, the patient is free from pain and no further thought is given to the tooth, the supposition being that it is all right. The case may go for years without involvement of the periapical tissues until possibly, without apparent cause, an acute abscess suddenly develops, or a chronic abscess may develop and gradually destroy the bone about the end of the root.

INFLAMMATION OF THE DENTAL PULP

ILLUSTRATIONS: FIGURES 308-315.

The pulp becomes inflamed from injuries or infections the same as other tissues of the body, but has not the same powers of recovery. It also suppurates quite commonly when it becomes inflamed. This suppuration is identical with suppurations in other tissues of the body, but the results of suppuration are especially destructive in this small bit of tissue, the more so because of its gelatinous type and its low degree of the power of resistance.

Etiology.

The most common cause of inflammation of the pulp is dental caries which has progressed sufficiently to involve the pulp or to expose it by laying it bare.

In decay of the teeth the dentin may be softened about a portion of the pulp tissue without the complete destruction of the dentin covering the pulp. It has been a favorite hypothesis among dentists that such softened dentin could act as a protection to the pulp, and it has even been incorrectly held that it would become hardened again. After medication, which has taken a pretty wide range from the strongest to the mildest antiseptics, a nonconductor has been placed over the softened dentin; or a nonconductor has been placed over it without other treatment. I have followed these forms of treatment very closely with records, and have found that the death of the pulp has generally occurred within a variable time, regardless of the treatment employed. This has been so general that I have repeatedly urged that the last bit of carious dentin should be removed, and then the pulp handled as might seem best.

In caries of the teeth, the acid-producing micro-organisms grow into the dentinal tubules after an opening has been made through the enamel. The acid always penetrates the tubules of the dentin in advance of the growth of the organisms, both following the length of the tubules progressively. The dentin is thus penetrated, until the pulp is finally reached. The acid softens the dentin, and these organisms and others, including those which produce inflammation and suppuration, and many

saprophytes*, which follow the progress of the organisms producing decay, all together melt down and destroy the softened tissue. Therefore, very soon after the softening of the dentin has reached the pulp, micro-organisms of various sorts may come in contact with the pulp tissue and infect it. This has been universal in the tissues I have prepared and brought under the microscope.

Inflammation of the pulp may result from its exposure in cavity preparation. When the excavating is done without sufficient regard for the depth of decay, or for the form of the protrusions of the horns of the pulp into the crowns of the teeth, particularly in the bicuspids and molars, many exposures will result. Generally a touch of any part of the pulp in excavating will be sufficient to set up an inflammatory movement which will cause its death.

The pulp may be exposed by breakage of the tooth, or other violence which crushes or destroys such portions of the hard structure of the tooth as will lay the pulp bare. Occasional cases occur in which the pulp dies as a result of a blow upon the tooth which does not fracture the tooth. The pulp evidently dies as a result of the injury to the tissues about the apex of the root. This occurs most frequently in the incisor region.

Pathological changes.

The result of inflammation of the pulp in the tooth of an adult is practically always the death of the organ. This has been referred to in a previous chapter in considering the healing powers of the pulp.

Generally in those cases in which decay has reached the pulp, but it is still covered by softened dentin, the pulp, upon microscopic examination, will show a small area of inflammation within the part of its tissue. (See Figures 308 to 312.) This area is particularly liable to be small if no symptoms have occurred. In cases in which symptoms have occurred, the area of inflammation will be found broader as the rule, although, as already stated, the symptoms are generally the same as those in hyperemia. In many of the cases in which very considerable pain had been invoked during the inflammatory process, I have found in the pulp tissue small abscesses, or abscessed cavities, which seemed to determine very great exacerbations of pain.

* I have used the word *saprophytes* to designate a class of micro-organisms which will not grow in living tissue, but may grow close about diseased parts and effect the decomposition of pus or other decomposible material, producing products which may or may not be poisonous to the living tissues.

(See Figure 313.) However, in a few rather widely inflamed pulps, giving the same symptoms, there was no evidence of abscesses when the pulp was examined microscopically. This makes the general correctness of the subjective sign of abscess formation uncertain.

Some years ago I had the opportunity to study one case of suppuration of the pulp; the patient refusing to have it removed. In this case, abscess after abscess occurred in the bulb of the tooth, which was a lower molar and easy of access. It is interesting to note the relation of pain to this abscess formation. In the first instance of lancing the abscess, the patient refused radical treatment, although he was in great pain. The point of a very sharp eye-knife was passed into it and a goodly drop of pus was discharged. This rendered the patient comfortable at once. In about a week he was again suffering and the same process was repeated with the same result. This was repeated five or six times within about as many weeks. The abscesses always caused intense pain which was relieved by the discharge of the pus. But this pulp was losing tissue continually during this time, until there was not very much of the bulbous portion left. Finally I had to reach into one of the roots to discharge an abscess occurring in that portion of the pulp. This might have gone farther, but the patient finally accepted extirpation of the pulp and the ordinary root treatment.

This I regard as a very interesting observation, and shows that great pain in the pulp of a tooth may be due to abscess formation. It will explain some cases which are very difficult to understand otherwise. For instance, one may have suffered very severe pain from the teeth, and finally that pain may have ceased almost instantly. It may recur later with a similar paroxysm lasting possibly several days, and then pass away. This seems to represent the formation and discharge of pus from these tiny abscesses in the pulp tissue. In my cuttings of pulps I have found a considerable number of these abscessed cavities, and this pathology and symptomatology coming together would be very interesting if we could make any especial use of them in directing treatment; but as these pulps will die anyway, we are not able to make practical use of the information gained.

Figure 312 is from a case of suppuration of a pulp showing hollowing out of the central portion. Such cases are not very frequent, yet the tendency of the suppuration to attack most prominently the central gelatinous portion of the pulp has become evident.

Diagnosis.

Pulp exposed to carious dentin. The one determining sign that inflammation has begun in the pulp is the finding, by careful removal of all decay, that the pulp has been exposed to carious dentin. If it has, it should be considered an inflamed pulp, whether it has given any symptoms or not, and treated accordingly. If, however, it is still covered by hard dentin, it should not be regarded as an inflamed pulp, no matter what the symptoms have been. I give this as the most certain way to determine clinically between inflammation and hyperemia of the pulp that I have yet been able to make out.

Pain. Pain is a very usual symptom of inflammation of the pulp. In very many cases, the progress of the inflammation is very quiet, producing but little pain. It is not uncommon for pulps to die from inflammation and suppuration, giving no symptom. This may occur whether the pulp chamber is open or closed. However, paroxysms of pain are liable to be induced by thermal changes, giving the symptoms of hyperemia. In every case of apparent hyperemia, it must be determined that the decay has not reached the pulp. This is necessary to make a proper diagnosis, which will determine the course of treatment.

In some cases, the pain may be so intense and of such continuous duration as to finally almost rob the patient of reason. In some rare cases I have seen the most pronounced examples of this. I will relate one case.

A woman was brought to the clinic of Northwestern University Dental School a number of years ago, to consult me in regard to persistent pain. In this case the patient had applied to her dentist with pain which she located in the second bicuspid in the lower jaw. The dentist found a cavity in the mesial surface of that tooth, which he prepared, and finding no exposure of the pulp, he placed a gutta-percha filling. The pain, instead of being relieved, became worse. At the next sitting he exposed the pulp and removed it, using cocain anesthesia, and placed in a dressing. The pain continued and became very excruciating. He waited several days, during which the patient suffered continuously, and then brought her to me in consultation.

The patient was very weak and nervous because of loss of sleep and continual suffering. I found a cavity in the distal surface of the first bicuspid with the gutta-percha filling in the mesial surface of the second bicuspid lying right against it. Cutting away the gutta-percha filling so as to see the distal surface of the first bicuspid clearly, I broke away the overhanging

enamel with a suitable instrument, exposing the extent of the cavity. This tooth was not sore to the touch, but in opening the cavity I caused a great increase in the pain, even though I had not touched the pulp with my instrument. I had, however, forced some debris into the cavity and this pressed on the pulp.

In view of the fact that this patient had suffered so long and so severely, and was still suffering intense pain whether any operation was proceeding or not, I determined that the best course was to relieve the pain then and there at all hazards. When the cavity was opened sufficiently, I selected a broach, and passing it down carefully along the wall of the pulp chamber, I thrust it to the apical end of the canal, and quickly withdrew it with a little twist, luckily bringing away the whole pulp. I at once prepared the cavity, laid in a dressing, and sealed it with gutta-percha. Before I had finished, the patient was asleep. Some of the young men carried her to a bed without awakening her, and she slept about five hours. After she awoke she expressed herself as being free from pain, and the pain did not recur.

This case gives as good an idea of the intensity of pain which may be induced by inflammation of the pulp, as any that I have been able to select from my practice.

Chronic inflammation of the pulp. Chronic inflammation of the pulp occurs in many cases, though they are exceptions to the most general rule. The pulp will sometimes become exposed and give no sign in the way of pain except when pressed upon by something forced into the cavity in chewing food. The patient learns to avoid such injuries, and goes on with comparative comfort, chewing his food on the other side of the mouth until, after some months or years possibly, the pulp will die either as a result of the chronic inflammation or an acute infection. Such cases are usually followed by alveolar abscess unless they have prompt attention.

Sometimes cases persist in a state of chronic inflammation for several years. Such cases do not respond to other than palliative treatment. The attempt to cover them over with any kind of a capping generally results in the speedy death of the pulp. Some exceptions to this rule are found, but they are not sufficiently frequent to be entitled to consideration. Figure 314 is from a case of chronic inflammation of the pulp.

Hypertrophy of the pulp.

Hypertrophy of the pulp occurs in a few cases of chronic

inflammation of the pulp, in which a considerable cavity in the tooth has occurred, making a broad exposure of the organ. Its tissues will swell and be forced out into the cavity of decay through the opening into the pulp chamber. This growth of tissue may enlarge until it entirely fills the carious cavity. (See Figure 315.) In cases in which the condition is maintained for some time, saprophytic micro-organisms will decompose and remove all the carious dentin around the portion of the pulp which is extruded into the cavity of decay.

Sometimes I have found this so complete that the dentin walls were left very hard and firm, the softened portion having been digested and removed by the saprophytic organisms. This occurs occasionally in large cavities that are widely open, but in which decay seems to have ceased because of the interference of saprophytic organisms, the growth of which was incompatible with the growth of the caries fungus. The softened portion of the dentin is removed, leaving hard, blackened cavity walls. The decay is stopped, at least temporarily.

Diagnosis. The diagnosis of hypertrophy of the pulp is very simple, for the cavity is filled more or less completely with a reddened, fleshy material, with which the opposing teeth often come in contact. It is easily seen, whether or not it completely fills the cavity. It often happens that the portion of the pulp exposed in this way becomes covered with epithelium which has been transplanted from the neighboring gingivæ and grows there the same as will epithelium planted upon a granulating surface.

This hypertrophied tissue is generally not painful, except when the patient bites something down upon it. One soon learns to avoid this, chewing mostly on the other side of the mouth. Therefore this unused side is apt to become unclean, and the gums more or less reddened and inflamed.

In the further diagnosis of this condition and the differential determination from a growth of the septal gingiva which may fill a carious cavity and have precisely the same appearance as this growth of pulp tissue, one may pass a thin, flat instrument into the subgingival space, close to the gingival line, and move it toward the occlusal, keeping it against the proximal surface of the tooth. If it may be passed out to the occlusal readily, without displacing or lifting any of the tissue, the growth is from the pulp. If the tissue is caught and lifted, the growth is from the septal tissue and not from the pulp, for in that case the septal tissue protrudes into the cavity of decay.

Treatment. If it is found to be a protrusion of pulp tissue, a very broad spoon excavator, the edge of which is sharp, should be passed down between the cavity wall and the growth, and then swept across under the tissue, keeping it close against the walls of the cavity, cutting the whole mass loose from that portion of the pulp within the pulp chamber. If this stroke is successful, the growth will all be removed. If not, the stroke will have to be repeated to loosen the remaining portion.

This cutting causes a slight pain and a profuse hemorrhage. The hemorrhage will stop in a few minutes and the blood may be washed away and the cavity inspected. The stump of the pulp remaining in the pulp chamber may then be treated for its removal the same as any other exposed pulp. It may be desensitized by cocain in the usual way, or destroyed by arsenic. Neither of these will act quite so promptly on such a pulp as upon one which has not been so long in a state of inflammation, but they do not generally give especial difficulty.

If the examination shows the growth within the cavity to be from the septal tissue, an ordinary silk ligature may be carried through the interproximal space to the gingival of it, and the ends brought up around the growth. The hypertrophied tissue may be lifted out of the cavity, and the ligature should then be drawn hard against the enamel of the next tooth and the hypertrophied tissue cut away with a single quick pull. This will partly cut and partly tear away the growth of tissue, which has filled the cavity. As this is done, the effort should be made to leave about the normal amount of septal tissue. One should be careful to so place the silk ligature that it will not bring away the entire septal tissue, as I have seen done sometimes. This would create an injury which would never fully recover, because the septal tissue would be too short to fill the space properly.

The actual cautery may be employed to remove either the hypertrophy of the pulp or of the septal tissue. If the wire is white hot, it will cause no pain, and the hemorrhage will be less than by the other method given.

These growths have no malignancy. In cases in which there is a hypertrophy of the pulp, the pulp itself should, of course, be removed. The growth of septal tissue into such a cavity is caused by the roughened walls of the cavity, and when the surface of the tooth is made smooth by a filling, there is no tendency to a regrowth.

CALCIFICATIONS IN THE PULP CHAMBER AND THEIR EFFECTS UPON THE PULP TISSUE

ILLUSTRATIONS: FIGURES 316-341.

In my writing in the American System of Dentistry, I gave very accurate descriptions of a large variety of the calcifications found in the pulp chamber, with illustrations, including those which grow upon the walls of the chamber and those which grow within the tissue of the pulp separately from the walls. As a description of the formations, that writing was quite sufficient. A number of forms were described which are very rare. We are now more interested in the effect of such growths upon the pulp and the conditions under which we may expect to find them, than in the varieties of growths found.

Classification.

I will, therefore, in this writing, limit myself closely to the effect of these growths upon the tissue of the pulp, classifying them into a few specific forms. A limited number of these, if not excessive in their growth, are beneficent in their effect, but nearly all of them are pernicious. They must be regarded as pathological. The forms, which I need mention, may be divided into two classes, and each of these may be again divided for identification:

Calcifications attached to the walls of the pulp chamber.

1. Growths upon the walls of the pulp chamber in which the tubules are continuous with those of the ordinary dentin — secondary dentin.

2. Growths continuous upon the walls of the pulp chamber; beginning as secondary dentin, but in which the dentinal tubules progressively disappear and the growth continues as a non-tubular, clear calcification.

3. Growths attached to the internal wall of the pulp chamber, which are nontubular, clear calcifications from the beginning. In any of these calco-spherites, or small nodules, which have previously formed free in the tissues of the pulp may occasionally be included.

Calcifications growing free in the tissues of the pulp, unattached to the walls of the pulp chamber.

1. Nodular formations, growing free in the tissues of the pulp, usually confined to the bulb of the pulp. These may or may not contain calco-spherites.

2. Fusiform calcifications, occurring in the root portion of the pulp. These are usually disposed with their length parallel to the length of the canal.

3. Jointed calcifications in the root portion of the pulp.

4. More extensive growths of calcific materials, which fill up the pulp chamber, sometimes including more or less of the contents of the canals, especially in the molars.

Personal investigations.

I have made a wide pursuit of this subject for a number of years, examining not only teeth which I was able to find in my own practice, but numbers which were sent to me by others. With each of these I was furnished a written description of the case. These were very largely teeth extracted in preparing mouths to receive artificial teeth, and often comprised from two or three to a dozen or more from the same mouth. Some were teeth extracted for other reasons, from persons of widely different ages. These teeth were cracked open, the pulp lifted from its bed, and some of them were examined after decalcification, and sections were made for microscopic study. A number of pulps, which presented many calcifications, were simply spread as well as possible upon a glass slide and a cover-glass laid on for observation with the binocular microscope, using low powers. In this manner of examination good views could be obtained of the character of the growths. I secured a very large variety of cases within the years which I devoted to this study. Practically all of these may be said to fall within the range of the seven groups named above.

This classification does not include all of the forms which may be seen. Each specimen presents some special points of difference, no two being exactly alike. The classification of a great variety of forms is of little value to the practitioner, since one generally can not make a diagnosis except by finding them after the pulp chamber has been opened. Some of the conditions can not be definitely differentiated without a microscopical examination of the pulp tissue.

In my examinations of these teeth, reference to the history of the patient accompanied the particular tooth, and symptoms which might indicate calcification of the pulp were sought for continually. Some of these were cases of abrasion and erosion, in which the filling up of the pulp chamber with hard material was sufficiently evident to the naked eye. There were also cases in which the teeth had been sensitive for a time and the sensi-

Fig. 316.

Fig 317.

Fig. 318.

Figs. 316 and 317. Teeth from the same mouth, showing erosion which had cut so nearly through one that part of the crown had broken off. In both the former position of the pulp chamber had been cut through, but had been previously filled with secondary dentin and the pulps were not exposed. Specimens from Northwestern University Dental Museum.

Fig. 318. Secondary dentin in case of extensive abrasion. The position of the former pulp chamber has been reached by the wear, but it had been previously closed over by the building of secondary dentin. Specimen prepared by Dr. H. A. Potts. Photomicrograph by Dr. F. B. Noyes.

FIG. 319.

FIG. 319. Secondary dentin: A, Margin of primary dentin, showing a few of the tubules continuing into secondary dentin. P, Pulp chamber. *Noyes.*

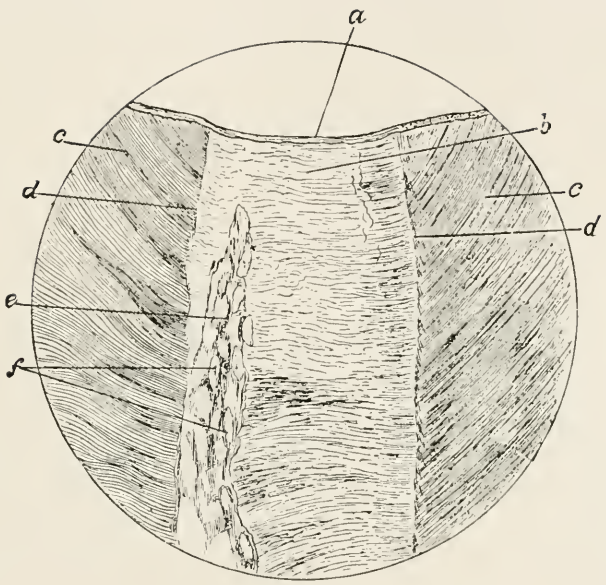

Fig. 320.

Fig. 320. Secondary dentin, magnified sufficiently to show the difference in primary and secondary tissue: a, Abraded surface of crown. b, Secondary dentin. c, Primary dentin. d, Junction of primary with secondary dentin. e, Remains of pulp tissue. f, Small oval masses of calcific material.

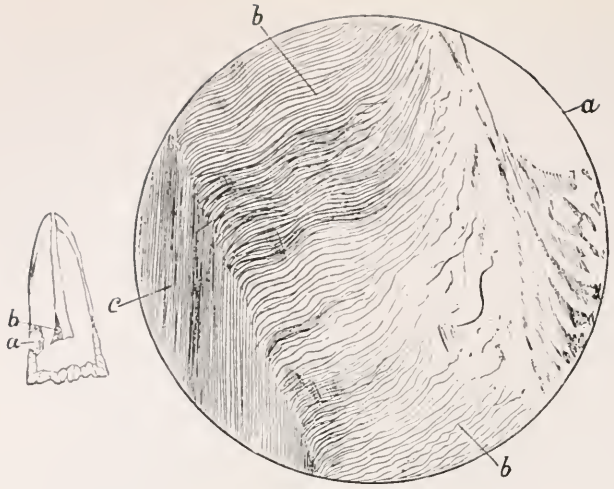

Fig. 321.

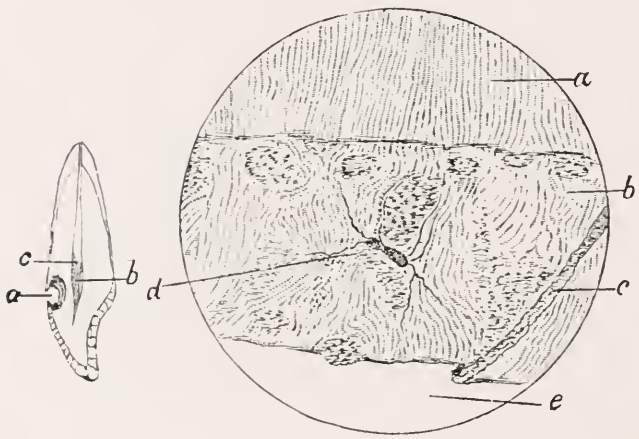

Fig. 322.

Fig. 321. Diagram showing deposit of secondary dentin, which was described in the American System of Dentistry, Vol. I, page 869, Fig. 464, as resulting from caries of an incisor. Caries at *a*, and secondary dentin at b. In the circle the structure of the secondary dentin is shown. a, Pulp chamber. b, b, Secondary dentin. c, Primary dentin. It will be noticed that the dentinal tubes in the secondary dentin gradually disappear, giving place to a clear calcification.

Fig. 322. Diagram showing secondary dentin, which was described in the American System of Dentistry, Vol. I, page 870, Fig. 465, as resulting from irritation of the dentinal fibrils by caries. Decay in the labial surface, *a*, and a deposit of secondary dentin, b. The point from which the enlarged drawing is taken is shown by c. In the circle the tissue of the secondary deposit is shown: a, Primary dentin. b, Secondary dentin. c, Seems to be a blood vessel that has become calcified. d, An irregular fault having some resemblance to the lacunæ of bone. e, Pulp chamber. It will be noted that there are irregular deposits of granular matter in the substance of the secondary dentin, and that the tubules wind about them.

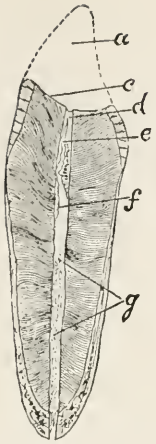

Fig. 323.

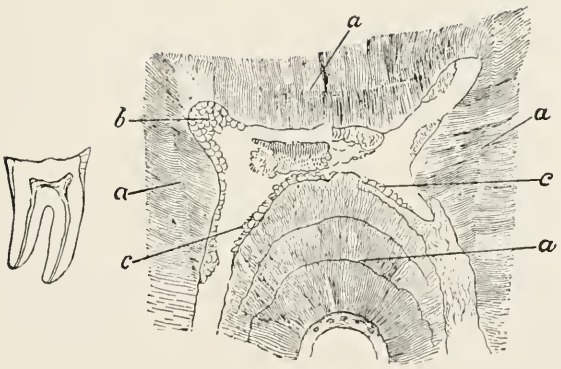

Fig. 324.

Fig. 323. Secondary dentin, filling the pulp chamber in case of abrasion of a cuspid tooth: a, Portion lost by abrasion. c, Abraded surface. d, Secondary dentin, filling a portion of the pulp chamber and acting as a protection to the pulp. e, Slender point of the pulp; irregular deposits are seen on the walls of the pulp chamber, as at f. g, Cylindrical calcifications in the root portion of the pulp chamber.

Fig. 324. Reduction of the size of the pulp chamber by deposit of secondary dentin as a result of abrasion. In the larger drawing the tissue of the secondary deposit is shown. a, a, a, Outline of the original pulp chamber, from which the secondary growth has begun; in the rootwise portion there appears a second line of beginning. b, Globular formation of dentin. c, Irregular crystalline deposit.

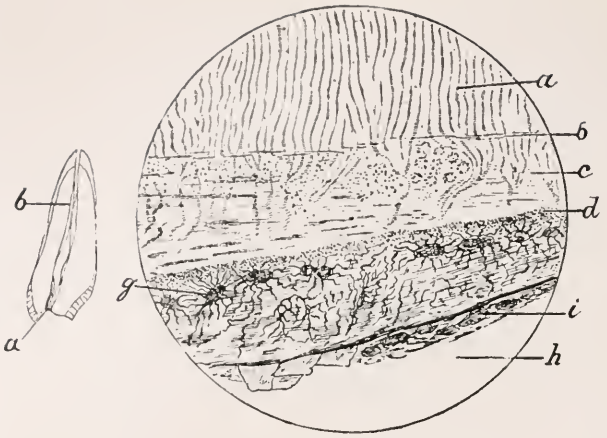

FIG. 325.

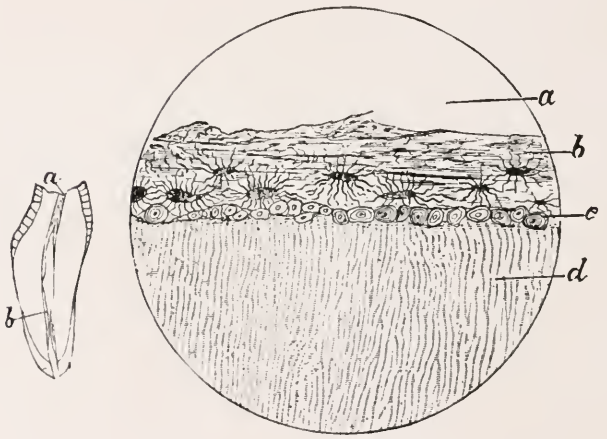

FIG. 326.

FIG. 325. Outline of incisor, showing a narrowing of the root canal at b by a deposit of secondary dentin. In the circle the structure of the formation is shown: a, Primary dentin. b, Line of the beginning of a growth of secondary dentin. c, Secondary dentin. d, Layer of granular matter. i, Irregular crystalline deposits. h, The pulp chamber.

FIG. 326. Outline of abraded incisor, with point of pulp chamber (a) closed by secondary dentin. b, Points out a narrowing of the root canal by a deposit of secondary dentin. In the circle the structure of the formation is shown: a, Pulp chamber. b, Calcific material. c, Layer of very small calco-spherites. d, Primary dentin.

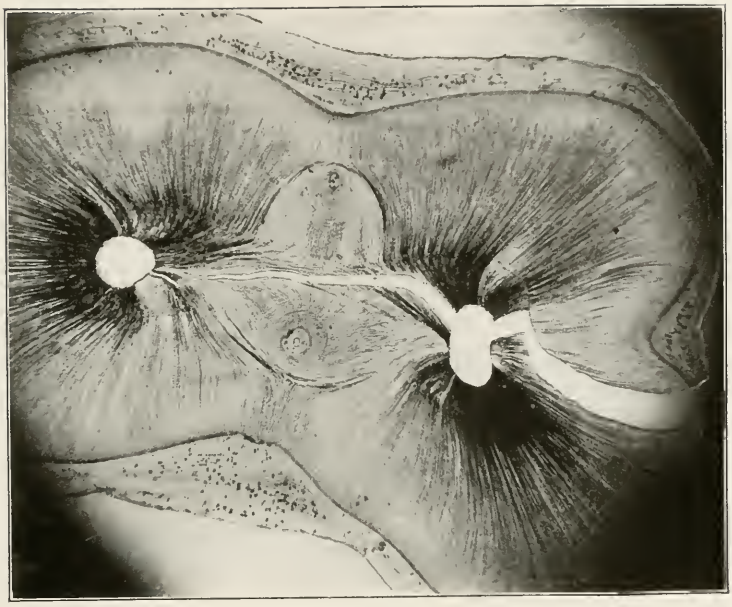

Fig. 327.

Fig. 328.

Fig. 329.

Fig. 327. A transverse section of a root, showing the reduction in the size of the pulp and formation of secondary dentin. *Noyes.*

Fig. 328. A central incisor showing extensive abrasion. The pulp is all calcified except a mere shred that shows as a white line.

Fig. 329. A central incisor, the greater part of the crown of which is worn away. The pulp is completely calcified far into the root.

Fig. 330.

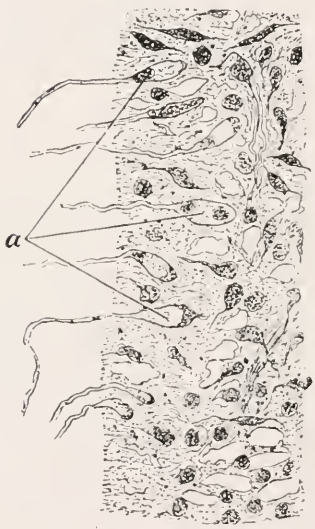

Fig. 331.

Fig. 330. Atrophy of the odontoblasts in connection with the building of secondary dentin. (Compare with Figure 297.)

Fig. 331. Atrophy of the odontoblasts: a, Odontoblasts that have taken the stain in an irregular manner. There is also a peculiar variation in their size. Some vacuolations appear in the tissue.

tiveness had disappeared; other cases which were still sensitive at the time of extraction, in which abrasion had proceeded only so far as to slightly expose the dentin. The examination included cases of extensive abrasion and erosion, as well as cases which had neither abrasion nor erosion. There was a wide variety in the ages of the patients. The study was practically exhaustive, and as I look back upon it, much of it seems to have been almost redundant.

Calcifications Attached to the Walls of the Pulp Chamber.

Under this heading those calcifications generally known as secondary dentin will be described. While these present the differences previously enumerated, they are so intermingled that it seems best to consider them together, as a single class, presenting the variations denoted in the groupings we have named. In the examination of a number of specimens, we will find some in which the tubules continue regularly into the new formation — true secondary dentin, others in which over a space the tubules are missing at the beginning of the new growth. (See Figures 319, 320 and 321.) This may occur in small patches. In another case quite a large proportion of the new growth will be nontubular, clear calcification, and in others complete cutting away of the tubules marks the beginning of the new growth throughout its attachment to the original dentin. The cases in which the tubules continue across into the new growth are very much more frequent than those which begin with a clear calcification.

In these growths more or less reappearance of dentinal tubules may occur in irregular forms, but they rarely straighten into regular dentin formation; much will be simply clear calcification. These generally are continuous upon the walls of the pulp chamber, but are found upon the walls of the root portion as well. The growth occurs on the root-wise portion of the double and triple rooted teeth, or in the floor of the pulp chamber, much the same as upon the occlusal portion and axial walls of the pulp chamber. In some growths, which begin at a single point upon the wall of the pulp chamber, a considerable process of almost any conceivable form may grow out into the pulp tissue. These are very generally clear calcifications, but sometimes they show a confused dentin formation with the dentinal tubules twisted among each other in vague and indefinite forms.

Etiology.

These calcifications occur under many conditions, most of

which are abnormal. They occur oftenest, and can be regularly found in cases of abrasion and of erosion, and it is in these that their causation and general history may be best studied, because they present a large variety of specimens, from those which are just beginning to those which have made wide progress. Figures 316 and 317 are of teeth which had been cut through the position of the pulp chambers by erosion. Figures 318, 320, 328, 329 and several other illustrations are of cases of abrasion. Something of the same character of secondary dentin occurs as a result of dental caries, if the decay involves a considerable number of teeth and progresses slowly, keeping the fibrils more or less exposed to irritation. (See Figures 321 and 322.) If caries progresses with what we may call normal rapidity, generally no deposit of secondary dentin will occur. Hence, in preparing cavities for filling operations, we generally do not find secondary dentin protecting the pulp.

In the summing up of the results of my observations, it has seemed clear that extensive abrasion of the teeth is in a degree hereditary. Therefore, the family history becomes of some importance, particularly in the matter of treatment.

Nature and conditions of growth.

The new growth begins upon the walls of the dentin, the tubules running across the line of the beginning new growth, but generally with enough of deviation of their course, or a reduction of the caliber of the tubules for a little space, to show quite distinctly the line where the new growth began. This regrowth may extend a considerable distance as fairly regular dentin. Some very peculiar features are discovered in extensive microscopic studies of these growths.

Protection for pulp. The physiological import would seem to be that the growth of secondary dentin is a response to irritation of the dentinal fibrils, and has a definite intention of placing the soft tissues of the pulp farther from the source of injury, and thus protecting it. From any viewpoint whatsoever, this idea stands out prominently. For instance, when we observe a tooth that has a number of facets worn into the dentin in cup shapes in the position of the cusps, and particularly in those cases in which the patient has had much pain from these in biting hard substances, because of the sensitiveness of the exposed dentin, we may know that the growth of this secondary dentin is starting.

Calcification more extensive as abrasion progresses. The

secondary dentin becomes more extensive as the abrasion progresses, and finally in cases in which most of the crown of the tooth has been worn away, a clear area of calcific deposit may be seen in the position previously occupied by the pulp. (See Figures 318, 323, 324.) This is different in color from the dentin surrounding it; usually it is a clearer variety of calcification.

Secondary dentin deposited through reflex action — not a local formation. The most general idea expressed in the literature has been that this secondary dentin is a local formation, confined mostly to the protection of the pulp over the area which is threatened by the injury to the dentinal fibrils. My extensive examinations show that the formation occurs reflexly from an impression made upon nerve centers by the irritation of the dentinal fibrils. This effect is general to the teeth of the person, and not localized to individual teeth; nor is it localized over the regions of the pulp especially threatened by the irritation of its fibrils. That is to say, it is not confined to local parts of the individual pulp chambers, nor to the teeth which have been worn, but occurs also in those teeth that may have escaped wear as a result of the previous loss of teeth of the opposite arch. Such teeth, though unworn, will show the calcification almost precisely the same as those which are worn.

For instance, I once received from a neighboring dentist twelve teeth extracted for one person, among which there were two molars that were unworn. He wrote me that with the exception of the wear upon the teeth, which had been extensive, the case had presented no abnormalities. I immediately wrote him, asking how it happened that those two molars had escaped wear. He returned the reply that the individual had had the molars from the opposite jaw extracted many years before.

I examined each one of these teeth individually, and found that the secondary dentin had started, as explained in the beginning of this description, as a fairly regular formation, upon the previously existing dentin; but it had begun in the same way and had proceeded to the same extent, in the teeth which were unworn. In the teeth which were worn, the pulp chambers, even in the molars, had been completely obliterated and mostly worn away. The pulp chambers of the unworn teeth were obliterated in the same way, showing that the effect was not local to the teeth worn, but involved all the teeth of the person. This I have found to be the rule in all of these extensive calcifications of the pulp.

In this particular case the wear had been quite a little different upon different teeth, and in some of them the second-

ary deposit had not all been worn away, which gave me the opportunity to examine critically the secondary dentin on what had been the floor of the pulp chamber. The fibrils passed fairly regularly into the new formation, but after they had passed into this for a space, they began to drop away more and more rapidly as we proceeded deeper into it, until the calcification became clear and free from tubules. It is this part of the calcification that is seen in the centers of pulp chambers which have been filled up, and have afterward been abraded.

In comparing these with teeth in which the formation has not been so extensive, we will find the beginning on the floor to be the same as the beginning upon the occlusal wall of the pulp chamber; the calcification occurring upon the walls of all parts of the pulp chamber together. (See Figure 324.) This could not be so if the calcification had been confined to the region of the pulpal ends of the dentinal fibrils that were irritated by the abrasion, proceeding from the occlusal surface of the tooth; or erosion proceeding from the labial or buccal surface. Hence, we are forced to the conclusion expressed, that all of this change has occurred through reflex action, and is not localized in any part of the pulp chamber, but is general to the pulp chambers and to the teeth of the individual. (See Figures 325 and 326.)

None of those who have reported investigations along this line seem to have made what I would consider extended examinations, involving a sufficient number of teeth, under the varied conditions under which calcification begins and its progress continues; consequently they have not had variety enough to give just conceptions of the beginning and progress.

I began my studies with the ideas derived from this literature, with the expectation of finding them correct. In some cases this seemed to be confirmed in observations of differences in the thickness of the growth in different parts of the pulp chamber. These differences were, however, discovered to be in the walls of the pulp chamber remote from the point of irritation, as well as upon parts over the fibrils irritated. Therefore, I feel that the descriptions in the literature, of this strict localization of deposits to the portions of the fibrils injured, has been too hastily assumed. Instead of examining as many as forty teeth, as expressed by Salter, it requires examinations of hundreds of teeth for reliable determination of this question.

I have thought that I could determine this matter more definitely if I could obtain teeth of children that had been so

broken at an early period, as to expose a large area of the dentinal fibrils, but in which the pulp had remained alive. Such a case would present an isolated exposure of the dentinal fibrils. I have thought to examine these, to determine whether or not a building of secondary dentin had occurred over the pulpal ends of those fibrils which were exposed to irritation. Opportunities for such examinations have, however, eluded me to such an extent that I have been unable to determine the point satisfactorily.

The effect upon the dentin and enamel. The effect upon the dentin of cutting off the fibrils from the pulp is to destroy its life. The secondary dentin may grow and narrow the pulp chamber without affecting the dentinal fibrils, but when the dentinal fibrils have dropped out, and a clear calcification begins, the fibrils in the dentin die. Those tubules which are exposed to the saliva become filled and soddened with the materials of decomposition which occur in the mouth, and in time the whole of this area of dentin becomes softer than normal. It can be cut without pain to the patient, though the line of demarcation as to pain is in many of the cases much broader than the exposure of the ends of the tubules would indicate.

The softening of the dentin renders the enamel much more liable to break away from it than from healthy dentin. This is a matter to be reckoned with in all of our operative procedures. This subject is considered in my work on Operative Dentistry. I will only mention here the natural consequences of the failure of the dentinal fibrils to pass regularly from the pulp through the dentin, and keep up the life which should exist in that tissue.

Abraded dentin becomes darker, fibrils die. The abraded dentin becomes darker than normal, causing the clear calcification to stand out more prominently in cases in which the wear has involved the positions of the former pulp chamber. When sections are cut centrally toward the pulp through the exposed fibrils, an area of darkened dentin will be discovered, which includes the fibrils of this exposed area from the enamel to the pulp. Examinations of teeth in the mouth will show that this area of dentin has lost its sensitiveness, or in other words, that the fibrils are dead. If one cuts beyond this line, the dentin will be found to be normally sensitive. In examinations of worn areas, as they may be discovered in the mouth, the color of the worn dentin will indicate whether or not the fibrils are alive. If the worn dentin is bright, or of normal color, the area will usually be sensitive and the fibrils will still be living. If the

worn dentin in the area is found to have become yellow, or darkened, it will not be sensitive because the fibrils will have died.

Exposure of pulp by abrasion and erosion. These calcifications act as a protection against the exposure of the pulp by abrasion or by erosion. It is a rare thing, which I have seen but a time or two in my life, that erosions have progressed rapidly enough to have exposed the pulp. They may go on and cut a tooth in two, allowing its crown to drop away, but before they have reached the pulp, the pulp will have been protected by a growth of secondary dentin, and the cutting proceeds through this secondary growth in removing the crown of the tooth. (See Figure 317.)

I have seen the pulp exposed very much oftener in what we may term the ordinary abrasion of the teeth, than from erosion. Generally this exposure in abrasion will be of the tips of the horns of the pulp, arousing first the symptoms of hyperemia, soon followed by an inflammation. This generally occurs in cases in which the horns of the pulp are unusually long, and the abrasion beginning upon the cusps exposes them. In these cases one may not be able to see an exposure of the pulp, but by taking a very fine broach and placing the point successively about the central part of the abrasion, where the horn of the pulp should be, the point is likely to drop into a very fine opening, which reveals the exposure.

Effect upon the pulp. While I have found very little in the symptomatology to indicate it, as a general rule I think it will be found that the injurious effect upon the pulp has been slower in cases in which the fibrils have crossed the line and entered some distance into the new formation.

I have cut sections of many teeth with the especial view of studying this point, in cases in which I have taken the symptomatology myself, or have had very exact expressions of it from those who had examined the cases. In so doing I found that the symptomatology did not aid me in determining what calcifications would be found. When calcifications have made considerable extensions upon the walls of the pulp chamber, narrowing it, there is a marked effect produced upon the pulp tissue. The cells of the pulp dwindle in size, and as the deposit becomes clear the odontoblasts disappear, and the pulp tissue simply lies against the growing clear calcification without the interposition of the odontoblastic layer. (See Figures 330 and 331.) The effect upon the pulp is more pronounced than in the cases in

which the calcification is tubular. Indeed, in the one case it is not properly secondary dentin at all, but a clear deposit; in the other it is true secondary dentin for a space, which gradually gives way to clear calcification by the thinning out of the dentinal tubules, with a corresponding dropping out of the odontoblastic layer. The effect upon the dental pulp deepens as the accumulation in the pulp chamber becomes greater. Many of the cellular elements disappear, or become mere threads, and the general expression given by the field under the microscope is that the tissue has become more distinctly fibrous until its structure is greatly changed. As this goes on, the pulp usually becomes insensitive and fails entirely to respond to temperature changes. The condition is a more or less complete loss of function by the pulp. (See Figures 327, 328 and 329.) Finally death of the remaining portions of the pulp occurs.

This is true of all kinds of calcifications which materially fill up the pulp chamber. A few calcifications scattered through the mass of the pulp tissue do not seem to produce this effect. In this consideration it seems that the only difference in effect between the calcifications beginning as clear calcifications, and those that begin as secondary dentin proper, is found in the suddenness of the interposition of clear calcifications to prevent the communication of the living pulp with the fibrils of the dentin.

Thus in the beginning, the effect produced by these calcifications is to protect the pulp for the time from exposure by extensive wear, or other injurious processes going on, which keep the fibrils in a state of irritation. This effect is beneficent, as it gives a nearly normal usefulness of the teeth for a much longer time than could otherwise occur. It also greatly lessens the chance of early inflammation and death of the pulp.

On the other hand, the amount of pulp tissue left in the root portion of the pulp chamber has become very small in most of these cases. The apical foramen is also reduced to the narrowest limits, yet I have never seen a case in which it was entirely closed.

These cases occur mostly in what we may term old age; or we have the conditions of old age to deal with in the teeth, although the person may not yet be old. There is the same narrowing of the pulp chamber, and the same narrowing of the apical foramen in the latter stages of these cases, which occur in the teeth of a very old person when normal conditions have existed until late in life. In other words, it is a premature clos-

ing out of the life of the pulp of the tooth. Any considerable extension of the calcification on the inner wall of the pulp chamber, or indeed within the pulp tissue, means the final death of the pulp. In most cases, this process extends over many years.

Danger of alveolar abscess. In these cases the conditions for the production of alveolar abscess would seem to be reduced to the minimum by the small amount of tissue composing the dead pulp, and the extreme narrowing of the apical foramen. There is a further consideration, that in the majority of these cases there is no infection introduced through an exposure of the pulp. This gives a fair assurance of continued health to the parts. It rarely occurs that such a pulp becomes infected through the blood stream by way of the apical foramen. As a matter of fact the formation of alveolar abscess is not common about the roots of these teeth.

In studying these cases with the microscope, we occasionally find a filament of the living pulp running far along to one side of the main body of the calcification. If the wear should open into this, as it sometimes does, we may be surprised by the sudden occurrence of an acute alveolar abscess. A number of such cases have occurred in my practice, and I made cross sections of the teeth, the pulp tissues of which were deeply calcified, and in several of these I found a fine opening running along the side of the calcification near one of the walls of the original pulp cavity which explained the occurrence of the abscesses.

Calcifications Growing Free in the Tissues of the Pulp, Unattached to the Walls of the Pulp Chamber.

Variety of forms. These calcifications take a multitude of forms. The most common are the nodular formations in the bulb of the pulp, ordinarily spoken of as pulp nodules or pulp stones. (See Figures 332, 333 and 334.) Next in frequency are the fusiform, or spindle-shaped, calcifications in the root portions of the pulp. In some cases the various calcifications in the root portions are joined together. (See Figures 335, 336 and 337.) Occasionally, all, or nearly all, of the tissue within the pulp chamber and root canals is found calcified, either in a single mass, or in several masses, more or less closely united.

On making sections for microscopical study, most of these calcifications are found to be homogeneous or clear calcifications. These are all nontubular. These sometimes have threads of tissue which are not calcified, irregularly mingled in their substance. In some specimens there are many of these, in some

very few, and in others none. What the physiological process of the growth may be in these clear calcifications seems not to have been made out. I have not found the onion-like layers which belong to calco-spherites. These are certainly not deposited after the fashion of the deposits of calcium salts in the building of the dentin, enamel or calco-spherites; otherwise we would find these layers a prominent factor, which we do not. Occasionally calco-spherites occur. These are the only definite forms found in the calcifications unattached to the walls of the pulp chamber. They will be considered more in detail in the following pages. In the bulbs of the pulps of molar teeth the calcifications may be round or irregular in their formation. If they are very irregular and nodular, they are apt to include a few calco-spherites and sometimes many of them.

Another very curious form is that in which the root portion is filled more or less completely with long spiculæ of hard formations which seem to be jointed, the ends resting loosely upon each other. These give a stiffened appearance to a pulp that is removed when in this condition, which has been called the lead-wire formation. Such a pulp seems stiff. You may bend it in any way and it will stay just as you have bent it, as would a piece of soft lead wire.

Sometimes we see pulp stones of conglomerate character that have filled up the entire bulbal portion of the pulp of a molar tooth without having any attachment whatever to the walls of that cavity. When we come upon them in the effort to remove the pulp, we find that they have a slight movement in the pulp chamber, showing that they are not attached to the walls. These curious forms may contain many calco-spherites; they may contain a few, or they may contain none. When a section is made we may find a clear calcification showing no forms, or it may have fibers running irregularly through it.

It is my opinion that these calcifications in the bulb of the pulp are less frequent than the calcifications in the root portion. Those in the root portion are fusiform calcifications in the main, with their length disposed parallel with the length of the root canal. They generally seem to have fibers of tissue attached to them, and especially to their ends, and sometimes they are considerably marked by fibers running through them, the forms of which seem not to be under any special control. There is nothing in them that we can consider as dentin, or the attempt to form dentin. They simply grow there, finally in such numbers as to strangle the tissues of the pulp and cause its destruc-

tion. This seems not dependent upon the same causes that bring about secondary dentin.

I have found nodules in the bulbs of pulps of teeth from children fifteen or sixteen years old. These, however, are somewhat rare. They are found oftener in the pulps of teeth of persons in or past middle life.

Generally no symptoms. There are some writings which represent the efforts of persons to diagnose the presence of these clear calcifications. Many have had the idea that the growth of these bodies was productive of some of the obscure pain found in regions about the mouth. In comparing my histories and my cuttings in cases where I have found these, I have been unable to trace them to any connection with such pain. True, I have sometimes found them in cases in which there was complaint of pain, but I have found them oftener in cases in which there had been none. Taking my studies as a whole, they indicate that the growth of these calcifications produces no particular symptoms.

The same is true of the growth of calcifications generally in the pulp tissue, or attached to the walls of the pulp chamber. They are all painless processes and give rise to no symptomatology, so far as I have been able to discover. They present difficulties in the removal of pulps, and in penetrating root canals, especially when the mass in the root canal portion has become very great so as to materially fill the canal.

Tendency to destroy pulp. Attention has been called to the fact that, while the deposit of true secondary dentin is for the time beneficent, yet when started, the growth continues to the destruction of the pulp of the tooth. This tendency to destroy the pulp is found also in the calcifications within the pulp tissue, especially those in the root portion. I think that a considerable number of these calcifications may continue in the root of the tooth for many years without doing apparent injury, provided their crowding out of that tissue is not too great.

The Treatment for Limitation of Calcifications within the Pulp Chamber.

The principal reason for presenting the subject of calcifications in the pulp chamber is to impress the importance of recognizing the relationship of abrasion and erosion to these formations, and of applying proper treatment sufficiently early to be effective in limiting their progress. This treatment must be prophylactic in its character. One must have in mind a clear

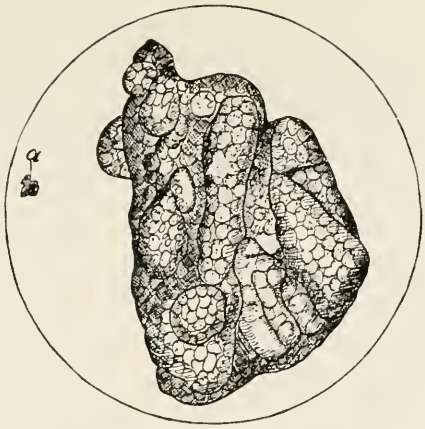

Fig 332.

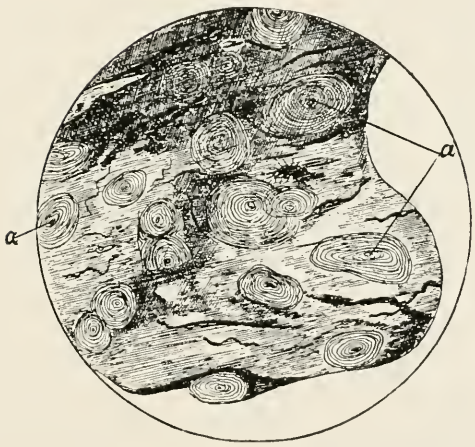

Fig. 333.

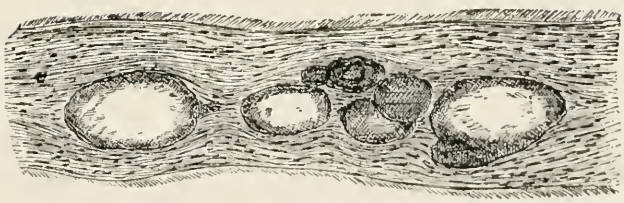

Fig. 334.

Fig. 332. A small pulp nodule as seen with a low power, showing its nodulation: *a* represents the actual size.

Fig. 333. Section of a pulp nodule showing many calco-spherites, as pointed out by a, a.

Fig. 334. Pulp nodules in the canal portion of the pulp.

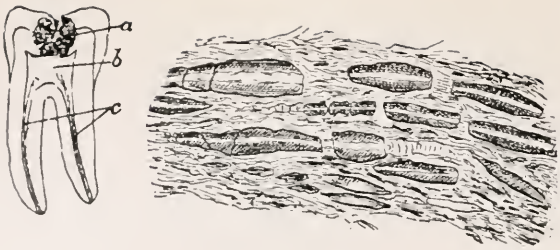

Fig. 335.

Fig. 336.

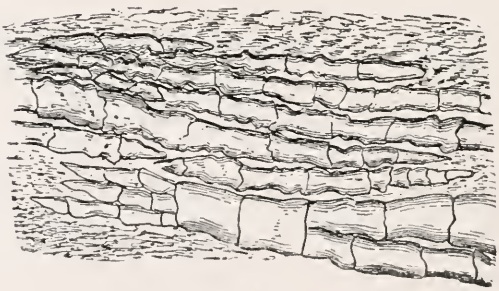

Fig 337.

Fig. 335. Outline of a lower molar with a large carious cavity at a. b, Pulp chamber. The shaded portion, c, was occupied by cylindrical calcifications. Sketch of the cylindrical calcification shown to the right.

Fig. 336. Cylindrical calcification of the pulp. This has been spread with needles, and the fibers that lay across the general trend show how the calcifications are attached at the end to the fibers. It will also be noticed that the tissue has lost its normal forms and degenerated into an irregular fibrous mass.

Fig. 337. Cylindrical calcification, more advanced than in Figures 335 and 336. Instead of running together and forming a solid mass, these are irregularly jointed.

conception of the conditions which induce calcification, and apply treatment for the removal or amelioration of those conditions. Treatment may be applied most successfully to abrasions and erosions of the teeth, also to a more limited extent to the calcifications which occur because of the exposure of the fibrils in caries of the teeth.

Treatment of abrasion.

In determining the plan of treatment to be followed inquiry should first be made as to whether or not other members of the family have had extensive abrasions of the teeth. The number of areas presenting and the extent of the wear should be carefully noted. This information will indicate the probability of success in the treatment. It is my judgment, from observation of many cases, that treatment may be employed with good effect in cases in which the areas presenting are small, and particularly if the patient complains of pain in biting upon the abraded areas. It is of importance to apply treatment very early in cases in which there may be evidence of hereditary predisposition.

In the treatment of the more recent abrasions, in which one or several teeth present with slight cupping of the dentin, cavities should be cut which will include each area, making such undercuts in the dentin beyond that which is exposed on the surface as will give good retention form. In the preparation of such a cavity, the dentin should be cut no deeper at any point than is necessary for the retention of the filling. The cavity should be filled with gold foil, or in preference, platinum-gold foil for the greater portion of the filling. I have found platinum-gold to wear a little better than pure gold, hence the recommendation of its use for these fillings. Another point of minor importance is that the color of platinum-gold is less objectionable, particularly in those cases in which several fillings must be made in the front teeth.

This filling should, in all cases, be made as hard as it is possible to make it by heavy malleting. In practically all such cases, the heavy use of the teeth in mastication has rendered the membranes firm and unyielding, so that heavy mallet pressure may be applied without discomfort to the patient. The cup that has been formed should be filled full and built out sufficiently to receive the occlusion of the opposing tooth upon its surface, and then trimmed just enough to render the occlusion reasonably comfortable. The intention should be to catch the occlusion

upon the fillings. If there are other areas in which the dentin has been exposed, they should receive similar treatment. The case should be followed by watchfulness and, as the dentin becomes exposed by the wearing away of the enamel at other points, fillings should be made.

As time passes, these fillings will become worn. The harder they are made in the beginning, the slower this wear will be, but in the general wear of the teeth, independent of this cupping process, the shallow fillings which are first made will wear out and other fillings must be substituted as rapidly as this occurs. The rule must be that the least possible abrasion of the dentin will be permitted.

In my practice this plan of treatment has been the most satisfactory. A number of cases have been followed long enough to establish this fact. This is true regardless of the question of heredity.

It must be understood that the patient is, in the beginning, to be impressed with the fact that no point of wear should be allowed to proceed after sensitiveness has occurred, but should be at once protected by a filling, and that this is to be followed year after year, as other points of wear occur.

This treatment will serve to protect the exposed fibrils and give them rest from irritation and, at the same time, avoid injury of the pulp tissue from calcifications. It will also effectively limit the abrasion of the teeth. This means a more or less complete prophylactic treatment against the occurrence of the evils resulting from calcifications.

Building up of extensive abrasions of the teeth. There are in the literature a number of papers dealing with the building up of badly abraded teeth by opening the bite sufficiently to restore approximately their normal length. I have studied this plan of treatment and its results in the practice of dentists, who have frequently employed it, and by the observation of cases that have been treated in this way, as well as by the teaching of my own attempts in this direction. Undoubtedly, this plan may be followed to great advantage when wear has affected a limited number of teeth, while the remainder occlude in such a way as to prevent wear. It should be understood that wear of the teeth occurs most in those cases where considerable lateral motion may be given to the teeth in chewing food, and least where the intercusping of the teeth prevents such lateral motion. In some mouths the lateral movement will be such that there is a considerable sliding motion of certain teeth in bringing the

teeth to the full intercusping in complete occlusion. These teeth may be worn, while others are not. If taken in time, the building up of the worn teeth may be of advantage, even though quite a little wear has occurred. For instance, it occasionally happens that the incisor teeth become badly worn and are becoming short, while the bicuspids and molars have not been much worn. In these cases such building as will limit the wear becomes important and the wear of these teeth may be delayed by the careful substitution of gold in such bulk as will not limit the occlusion. It may be possible to hold a sufficient proportion of such teeth for many years, if not permanently. Other cases simulating the above, where some teeth wear and others do not, are often susceptible to this treatment. By making careful selections of cases, much may be done in protecting teeth and also in protecting the pulps from calcifications.

My experience and that of others gives a sharp warning against undertaking the building up of extensive abrasions of the teeth. When great numbers of teeth have been built up, opening the bite, some of the operations usually fail within a few years, from one cause or another. Most of these failures occur from breakage of the teeth, so that the fillings become loose. As time passes, more and more of this breakage occurs. This is due doubtless in the main to a want of appreciation of the fact that the dentin is softened and that the whole of the enamel supported by the softened dentin is much more brittle than normal. The filling, therefore, has not an anchorage that is sufficient to sustain it against the heavy stress brought upon it. In studying these breakages, I have found that the anchorage in most of them was as secure as it could be made, and have come to regard the failures as unavoidable because of the existing conditions.

Danger of approaching too close to pulp in preparing cavities. Another condition has arisen in a number of cases that have come under my observation, which seems difficult to avoid. In seeking the best possible anchorage for fillings in such worn teeth, one is very likely to approach the pulp too closely, inducing the premature death of the pulp. Such a result is particularly distressing.

Treatment of erosion.

I have thought that something might be done with a view to limit the injury to the pulp tissues, by the calcifications which occur upon the walls of the pulp chamber in erosion. I have

tried this in a number of cases, but not sufficiently to have formed a good judgment as to its value. Such treatment was applied to cases in which the cutting was of definite wedge form in the gingival third of the labial surfaces of the incisors and cuspids.

If a cavity is prepared and a filling placed when the erosion has proceeded so far as to cut through the enamel, exposing the dentin, the depth of the erosion will be limited and further irritation of the dentinal fibrils will be prevented. Generally, however, this will not limit the breadth of the erosion upon the surface of the tooth and, unless the cavity is cut wide in the beginning, the erosion will soon begin along the margins of the filling. This will call for a new filling, wider than the first. In many of the erosions this plan might be followed until the widening of the erosion ceases; that is, until the erosion has reached what would be its full breadth.

This plan of treatment is encouraged by the fact that the erosion is liable to stop at any point in its progress and not again become active. If so, we will have made effective prophylaxis of the injury to the pulp by this treatment. This plan may be applied to the wedge form of erosion with better probability of success than to any other, as the wedge forms are inclined to cut deeply, without much spreading on the surface. It should not be attempted in any case in which the erosion is inclined to make a very broad shallow cut.

The Formation of Calco-spherites.

The term calco-spherites is applied to calcium deposits arranged in concentric spheres. The formations of calco-spherites are, I think, less common than the reading of our literature would indicate. They are found only occasionally among the calcifications which occur in the pulp tissue, but are often prominent objects when they are found. These are very peculiar and characteristic growths, having markings similar to those of a tiny onion cut across equatorially — that is, considering the top of the onion one pole and the root the other pole, and cutting it across the center between the two, which shows the markings most regularly around it. The calco-spherite resembles this more in appearance than any other object I know in nature.

We know very little positively of the manner of the formation of the calco-spherites. The curious experiments of Rainey and Ord, which have been repeated by others, including myself,

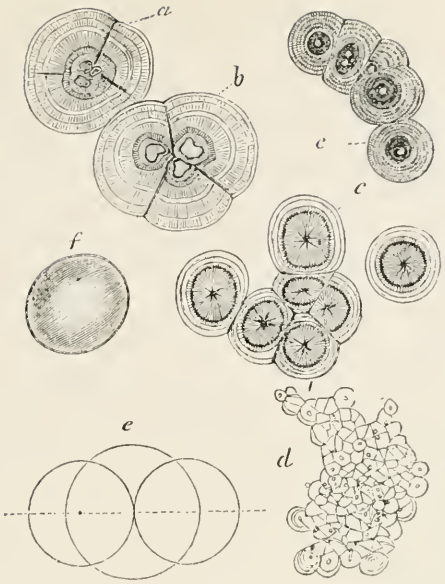

Fig. 338.

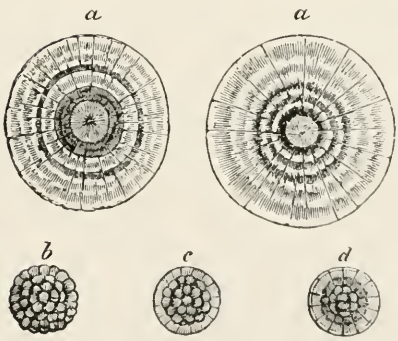

Fig. 339.

Figs. 338, 339. Artificially formed calco-spherites from book entitled: "On the Mode of Formation of Shells of Animals, of Bone and of Several other Structures, by a Process of Molecular Coalescence," by George Rainey, M. R. C. S., 1858. Fig. 338 "represents calculi as they are found on the under surface of the slides." Fig. 339 "represents one form of globule sometimes found both in the deposit and on the slides, as well as the most perfect forms of the laminated calculi with radii." (See reference in text.)

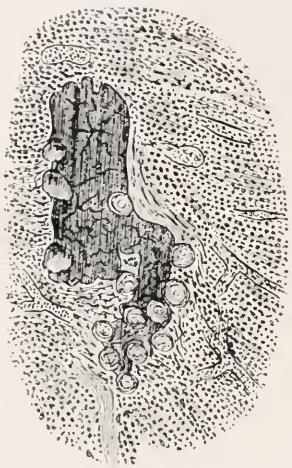

Fig. 340.

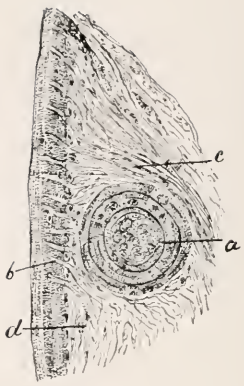

Fig. 341.

Fig. 340. Deposit of unusual form within the tissues of an inflamed pulp of a tooth of a child of 14. This deposit was soft enough to be readily cut with a knife.

Fig. 341. Calco-spherite-like spherule in the tissues of the peridental membrane: a, Spherule. b, Cementum, showing the fibers of the peridental membrane springing from it. c, Principal fibers of membrane. d, Indifferent tissue. For a small space no fibers are attached to the cementum.

have drawn general attention to the calco-spherites, and to their composition. Calco-spherites may be produced artificially by taking a solution of albumen, or an albuminoid substance in water, to which is added a small amount of finely pulverized calcium salts. The solution should then be impregnated with carbon dioxid under pressure. This should be sealed and set aside in a still place for several months, at the end of which time it will be found that calco-spherites have formed and fallen to the bottom.

In the body we are most likely to find calco-spherites in positions in which there has been stagnation of the blood, as in cases of phleboliths found in varicose veins.

THE ARTIFICIAL FORMATION OF CALCO-SPHERITES.

A book by Rainey was published by John Churchill in London, in 1858, in which he described minutely a method for the artificial formation of calco-spherites and gave numerous drawings to illustrate the various steps in the formation of these calculi. This book was published during the same year as Virchow's work on cellular pathology, which finally convinced the world of the correctness of the cell theory. Rainey unfortunately denied all possibility of cellular elements in life force, which at the present time naturally detracts from proper consideration of other questions discussed by him.

Rainey's book contains 152 pages, closely printed. The sentences are very long. In many respects the book is a curiosity. It is occupied with a detailed account of the author's conception that the shells of the mollusca, in their formation, are practically identical with the formation of the calco-spherites, which is described. He ascribes the same principle discovered in the formation of the calculi to the formation of bone in the lower animals and in man. I have had two of the illustrations in this book reproduced for this publication. (See Figures 338 and 339.) In his description he gives the following as a plan:

"This process is given in the Transactions of the Microscopical Society, published in the Quarterly Journal of Microscopical Science for January, 1858. It consists of introducing into a two-ounce phial, about three inches in height, with a mouth about one inch and a quarter in width, half an ounce by measure of a solution of gum arabic saturated with carbonate of potash (the subcarbonate of the old pharmacopœias). The specific gravity of the compound solution should be 1.4068, when one ounce will weigh 672 grains. This solution must be perfectly

clear; all of the carbonate of lime which had been formed by the decomposition of the malate of lime contained in the gum, and also all the triple phosphate set free by the alkali, must have been allowed completely to subside. Next, two microscopic slides of glass, of the ordinary dimensions, are to be introduced, with the upper end of one slide resting against that of the other, and with their lower ends separated as far as the width of the phial will permit; and lastly, the bottle is to be filled up with a solution of gum arabic in common water, one ounce of which will weigh 520 grains. This solution must also be perfectly clear, having been first strained through cloth, and then left to stand for some days to allow of the subsidence of all the floating vegetable matter. It must also be added carefully to the alkaline solution, that the two solutions may be mixed as little as possible in this part of the process. The bottle must now be kept perfectly still, covered with a piece of paper to prevent the admission of dust, for three weeks or a month. Time would be saved by having a dozen bottles thus charged, and examining their contents at stated intervals, according to the chief object sought for in the experiment. The soluble salts of lime to be decomposed by the subcarbonate of potash are contained in the gum, in combination with malic acid, and also in the common water; ammoniaco-magnesian, or triple phosphate, is also contained in the gum, and is set free by the alkali. Muriate of lime, dissolved in a solution of gum from which all the lime had been previously separated, would answer a similar purpose, provided the muriate were not in too great excess for the gum, in which case crystals of carbonate would be formed together with the globules, and the surface of the slide would become covered with coalescing patches of the latter. Also muriate of barytes, and muriate of strontia, when treated in the same manner as the muriate of lime, furnish each a globular carbonate, the spherical form of the latter being particularly perfect and beautiful. But muriate of magnesia, when decomposed in the same manner, and under precisely the same condition, does not furnish globules, but crystals of carbonate of magnesia, evincing no tendency to become globular."

To follow his methods further for the formation of large calculi, such as are seen in the hard secretions of the pulps of the teeth, would take up too much space to be included in this volume. These are described as the meeting of two calculi, which are drawn together by the mutual attraction, and the blending of two into one. These are very hard bodies and the

blending comes through disintegration and reformation of the two into one body. Others are added in the same way and the calculi become larger and larger. Each new addition blending with the calculi previously formed, but forming a ring around the calculi until many such rings may be seen, as in the calculi we sometimes see in the pulps of teeth.

The descriptions of these artificial formations occupy about half the book and the other half is devoted to a description of the principles illustrated in the artificial production of these calculi to the formation of shells of the mollusca, of the bones of lower animals, and of man.

Authors often mention in their reference to Rainey's book the name of Ord. I have found but one paper on this subject written by Wm. Ord and that is occupied entirely with descriptions of spherites formed from cholestrin. This article of eight pages appeared in the Proceedings of the Royal Society of London, June, 1879, p. 238. He describes bodies of similar appearance that are formed from cholestrin found in the gall bladder and in tumors of various kinds, by a treatment closely similar to those described by Rainey for calculi. This paper by Ord seems to be not only a careful and close description of these processes but it is worth any one's while to read it as a bit of scientific literature. He mentions experiments by Professor Guthrie, who formed very similar bodies from the salts of copper, which have the appearance of true copper, but are in fact a combination of copper and a colloid substance. None of these formations are true crystals. A crystallization seems to defeat entirely the result sought. This crystallization may result from impurities of the chemicals used and various other accidents. It is found that the addition of magnesia even in small quantities causes a crystallization instead of formation of calco-spherites and no calco-spherites seem to be formable in a fluid which contains magnesia.

Importance of studies of calco-spherites.

This fact will probably become important in the studies of what have been called "mottled teeth," in which there is a failure of formation of the cementing substance of the enamel, often accompanied by a discoloration of the areas from which the cementing substance is missing. These mottled teeth have been found in the mouths of as high as eighty-five per cent of persons living in certain localities during the period of the formation of the enamel.

When we consider that as a matter of fact the teeth and bones are formed by combination of calcium salts with colloids, from which we may dissolve the calcium salts, leaving the colloids in the soft state, we see the widespread importance of a closer study of this subject. There is no true crystallization in the formation of these calco-spherites, or, so far as I am able to grasp it, in the formation of the bones, dentin or enamel. In the calco-spherites the polaroscopical appearance is limited to the formation of a dark cross which is very characteristic of these bodies. The formation of indefinite bright spots and limited indefinite lines will also be found in the cut specimens of calco-spherites, dentin and enamel; and to a more limited degree in the bones, but there is no true polarization with decompositions of light, as will be seen in many of the true crystals. There are crystals which will decompose light into its component colors and crystals which will not do so.

I may say that I have been attracted to the study of these old experiments because, as has been stated previously, globulin already loaded with calcium salts (calco-globulin in fact) comes into the mouth with the saliva, and is formed into salivary calculus, and also globulin that has apparently no calcium salts in its make-up comes into the mouth and is not formed into salivary calculus.

To my mind, Rainey's book is a study of one such combination of lime salts with colloid material, and Ord and others who have written along the same lines, have presented a few other similar combinations. The chemical tendencies to the combination of these for the formation of hard substances from salts and colloid material should not be ignored in our study of these processes. These chemical processes must be regarded as under the control of the cellular elements, which determine the specific forms to be produced. The calco-spherite is formed when the elements are brought together and by some accident have fallen out of this control and are left to the chemical affinities of the substances uniting.

When we turn our attention to the life force as a controlling entity in the production of animal bodies, we must at once concede that the material coming under this influence is controlled in its chemistry in the main by the manipulation of these life forces, and material is withheld or added in proportion to the substance which the life force indicates. Hence, we get the extremely hard enamel or the softer bone, or the dentin lying between, and various other hard substances formed in this way.

In this view of the case the calco-spherite would seem to be in some degree related to a cyst, hedged about by a membrane, which develops for the purpose. The calco-spherite, however, has no such membrane that has yet been discovered, but lies among the tissues separate from control of the life forces which act upon these materials in the formation of definite tissues. Therefore, this material is left to form its chemical affinities uncontrolled by the life force. This play of chemical affinities was the particular thing which Rainey studied; other things were incidental.

This consideration of the subject would open a very wide field for discussion, but an expansion of this would be somewhat foreign to this book. There are some things which I have mentioned, as have others, indicating that the colloids are the agents used in the building of bone and very hard substances, or that calco-globulin, in the form in which it exists in the tissues, is such an agent, supplying both the hard material and the soft. These are manipulated under the influence of life direction in or among the cells which accomplish the change.

I have, myself, seen many very small spherules lying between the odontoblasts and the forming dentin in sections taken during the development of the teeth. Andrews, in his article in the American Text-Book of Operative Dentistry, p. 70, describes certain minute glistening bodies, occurring about the cells of the ameloblastic layer of the enamel organ. My interpretation is that these are in fact primary spherules of calco-globulin, of unusually small size. They are certainly not calco-globulin in the sense of calco-spherites known in the hard formations in the tooth's pulp. The naming of them by Dr. Andrews as calco-spherites seems to me to be a mistake. They are probably properly distinguished as calco-globulin.

Figure 340 illustrates an unusual formation found within the pulp tissue; Figure 341 a calco-spherite-like formation found in the peridental membrane.

TECHNIC OF TREATMENT OF THE DENTAL PULP

ILLUSTRATIONS: FIGURES 342-401.

IN my work on Operative Dentistry, I presented only the technic of exposure and removal of the dental pulp, and filling of root canals. In reviewing that writing at this time, I find practically nothing which I desire to change. I have, therefore, decided to include most of it, with the illustrations, as it appears in the Operative Dentistry, adding that which seems necessary to a complete presentation of the subject of pulp treatment in this book.

GENERAL CONSIDERATIONS.

There are certain general propositions which are common to almost all operations which involve the opening and treatment of pulp chambers and root canals. These will be presented first. In this connection, the dentist should hold foremost in mind the fact that the tissue of the pulp can not be considered as separate and apart from the other tissues of the body; that, in the matter of transmission of infection, the pulp bears the same relation to the general system as do other tissues. Therefore, in the treatment of the pulp, surgical cleanliness is quite as important as in the treatment of other parts. The field of operating should have the same consideration as to asepsis; all broaches, other instruments and dressings should be sterile; the dentist has no more license to use an unsterile broach than has a surgeon to deliberately operate with an infected knife. The far-reaching, serious and many times fatal consequences of infections entering the system through root canals demand that we give our patients the same protection which they receive at the hands of the surgeon. The technic for asepsis which will be presented is simple; it may be easily carried out to the last detail by every practitioner.

We should always approach the treatment of a pulp with a full appreciation of the fact that the continued usefulness of the tooth is dependent upon the success of the effort. Thoroughness in this class of operations is absolutely essential to success.

A good knowledge of the anatomy of the teeth and a large store of patience are necessary to thoroughness. If there is a single class of operations in dentistry which deserves the most conscientious efforts of which each operator is capable, it is the treatment of root canals. The knowledge that an improperly treated canal may not give serious trouble for many years has doubtless led to much carelessness and even recklessness in this operation. The advent of the X-ray has shown this very positively. Now that we are confronted with the fact that each error in this technic may endanger the life of the patient, it is imperative that every dentist should devote whatever of time and study may be necessary to gain the highest possible degree of efficiency.

Thoroughness in root canal treatment requires time. It often requires that the operator be in the best possible physical and mental condition. While one may not always be able to control the time of doing these things, they should generally be set for the first morning appointments, particularly for the final inspection of the canals and the placing of root fillings. Whenever the operator is uncertain of the conditions within the canal, either as to the removal of the last remnant of pulp tissue, or the probability of getting a filling well to the apex, a dressing should be sealed in and the patient given another appointment. In the more difficult cases one should not continue at any sitting if he feels that he is not making progress.

One other word which applies to all pulp treatment. We have not been sufficiently careful in educating the public to a proper appreciation of the difficulties presented in this service, the niceties of technic required, and the importance of it to the future usefulness of the teeth and to the general health. These are things which our people should know; which they must understand, if we are to have their coöperation to the end that we may take the time necessary to do the operations in the most painstaking manner, and that we may be properly and willingly recompensed for such service.

Asepsis.

The first rule of modern surgery is asepsis. As applied to pulp treatment this means that the field of operation should be treated as though it were a surgical wound. The immediate neighborhood should be maintained in an aseptic condition during each operation. Nothing carrying infection should be permitted to enter this field. All instruments and dressings should be unquestionably sterile. In no case should saliva be allowed to

enter a pulp chamber from the beginning of the first treatment until after the root canals are filled. This may be done by so simple a technic that there is no reason why it should not be carried out to the finest detail, except in a very limited number of cases which present unusual difficulties.

Plan for aseptic technic. It should be recognized that it is impractical, although not impossible, for the dentist to keep his hands surgically clean during pulp treatments. However, a safe technic may be employed by which ordinary cleanliness of the hands will be sufficient in most cases. In other words, a plan may be carried out by which the operator's fingers will not touch anything which actually enters the pulp canal, or the aseptic field, not even with the cotton wrapped on broaches. Under this plan, asepsis in pulp treatment requires: (1) That the rubber dam be applied for every treatment, and in such manner that there will be no leakage of saliva; (2) that the field of operation — all teeth included in the rubber, and the adjacent rubber — be rendered sterile by swabbing with an antiseptic before the pulp chamber is opened; (3) that all broaches, burs, excavators and dressings which enter the pulp chamber shall be sterile; (4) that the fingers be surgically clean, if they come in contact with cotton which is to enter the canal, or the aseptic field.

Application of the rubber dam. It would seem that no argument should be required to convince any thoughtful person of the absolute necessity of applying the rubber dam for the purpose of maintaining asepsis while a pulp chamber is open. It is necessary to keep the saliva from entering a canal, not only because the saliva is loaded with many varieties of microorganisms, but also because it contains material which will cause the discoloration of the tooth if permitted to be absorbed into the dentinal tubules.

It is just as important that the rubber be applied in cases presenting with the pulp dead and the pulp chamber widely open, as in cases in which the pulp is not exposed. The fact that the pulp tissue is already infected is not a reason for omitting to apply the dam. There is the same danger in treating such a tooth without applying the rubber dam and following rigid rules of asepsis as there would be if a surgeon should use unsterile instruments and dressings in an infected wound. As will be pointed out later, many chronic alveolar abscesses are caused by failures to observe the rule relative to the application of the rubber dam. The rubber dam should remain in place at each

sitting until after the cavity in the tooth has been securely sealed.

Sterilization of field. After the rubber is in place, the field of operation should be rendered sterile by swabbing the crowns of the teeth included in the rubber, also the adjacent rubber, with an antiseptic, such as oil of cloves. Then the field may be dried with alcohol. This should be done on each occasion before the pulp chamber is opened. If a temporary filling has been placed at a previous sitting, it should not be removed until the rubber is on and the field sterilized. If the cavity is a proximal one, a thin saw may be carried past the contact, thus trimming off enough of the temporary filling to permit the application of the rubber, without disturbing the filling.

Sterilization of instruments, dressings, etc. The thorough sterilization of all instruments entering the pulp chamber may be easily done by using two small dishes of suitable size, one containing 95 per cent phenol, the other absolute alcohol. The accompanying illustration, Figure 344, shows a special dish designed for this purpose, containing one depression the proper size for the immersion of a broach, bur, or other instrument, in phenol, and the other to hold a sufficient quantity of alcohol. The broach may be fully immersed in the phenol for a few minutes, then picked up with the pliers and washed in alcohol. After it has been placed in the broach holder, the end of the broach may be dipped into the phenol and washed in the alcohol as frequently as may be desired during the progress of the operation. Burs and excavators may be sterilized in the same manner. Gutta-percha points should also be immersed in the phenol and alcohol after they have been attached to the root canal plugger.

Sterilization of broaches wrapped with cotton. To avoid the necessity of having the fingers surgically clean in order that a broach may be wrapped with sterile cotton, the cotton should be wrapped upon the broach and then sterilized.* To do this without inconvenience requires that one have a simple dry sterilizing oven, and all-metal broach holders which will not be injured by the heat of the sterilizer, in sufficient number that a few will always be ready. Such a sterilizer is shown in Figure 342. This is an oven made of asbestos board, fitted with a single sixteen-candle-power lamp to heat it, regulated by a thermostat

* So far as I know, the first publication of a plan of sterilizing broaches wrapped with cotton was in a paper entitled, "The Surgical Treatment of Pulp Canals as a Prevention of Systemic Disturbances," by Dr. Elmer S. Best, in the Dental Review, Vol. 29, 1915, p. 320.

similar to that described in Figures 173, 174 and 175, for the warm-water tank. A thermometer registers the temperature. This oven is provided with porcelain trays, each of which holds six broaches; also dishes for cotton. (See Figure 343.) Such an oven may be made of sheet metal, and it is not necessary that the temperature be controlled by a thermostat. One may take a small metal box, place a lamp inside, and after a little experimenting, determine the time required for sterilization.

The oven referred to will hold twenty-four broaches, in four trays, also several dishes for cotton pellets of different sizes. Cotton is wrapped on the broaches, they are placed in the trays and these are placed in the sterilizer. The temperature of the oven is kept at 160°. The current is not turned off at night, but is left on, except as the thermostat disconnects it. Barbed broaches are not placed in the sterilizer; they are immersed in phenol and alcohol, as previously mentioned.

When a pulp treatment is undertaken, one of the trays containing six wrapped broaches and one or two dishes of cotton pellets of different sizes are placed on the operating tray for use. Afterward those not used are returned to the oven. Figure 343 shows the operating tray with the various dishes, instruments, etc., laid upon it. Figures 345 and 346 illustrate a very convenient dropper bottle.

Technic of wrapping cotton on a broach. If it is desired that the cotton adhere to the broach, a few shreds should be pulled between the fingers until there are but a few parallel fibers. One end of these should be held between the forefinger and thumb of the right hand and with it the broach should be grasped at midlength, or with a cotton wisp extending slightly past its point. With the left hand the other end of the cotton wisp and the point of the broach should be grasped together, and the broach rotated in the fingers of the right hand until the cotton is wrapped firmly upon its shaft. When this is properly done, the cotton will cling firmly to the broach and is not likely to be lost in the canal.

If it is desired to place a dressing in the canal, a wisp of cotton should be formed with its fibers mostly parallel, and the end of this caught with the point of the broach with the thumb and finger of the left hand and the broach rotated with the right hand, while the fingers of the left roll the cotton on its end. In this way the cotton is rolled on the broach in such a way that it will not slip backward on the broach and can be carried to the

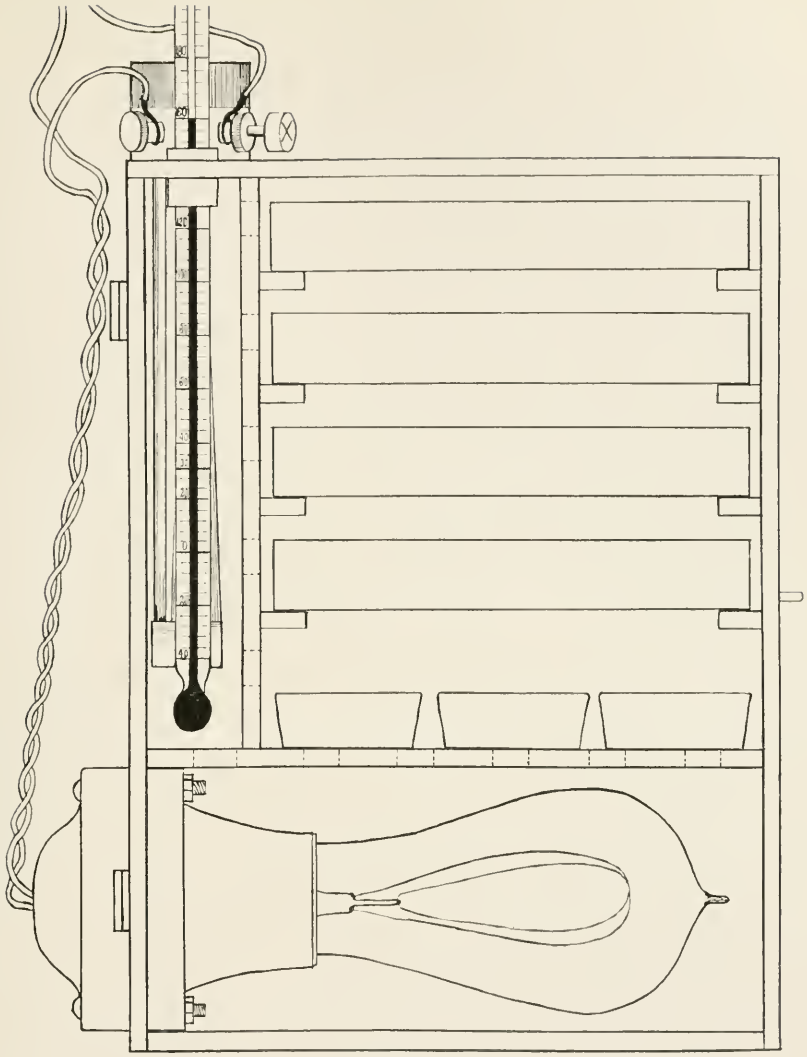

Fig. 342.

Fig. 342. A small dry sterilizing oven heated by an electric lamp, controlled by a thermostat. This oven holds four porcelain trays, each containing six broaches in the handles. Cotton is wrapped on the broaches before placing them in the sterilizer. There is also room for other small trays containing cotton pellets, etc. This oven is kept at 160° and the material to be sterilized is left in the oven several hours before it is used. One of the broach trays and the little porcelain trays for cotton pellets are shown in Figure 343. The thermostat is illustrated in Figures 173, 174, 175.

This oven is made of $\frac{3}{16}$-inch asbestos board, and its actual size is $6\frac{3}{4}$ inches wide, $9\frac{3}{8}$ inches high, and 3 inches deep. The sketch is one-half actual size. The door is not shown.

FIG. 343.

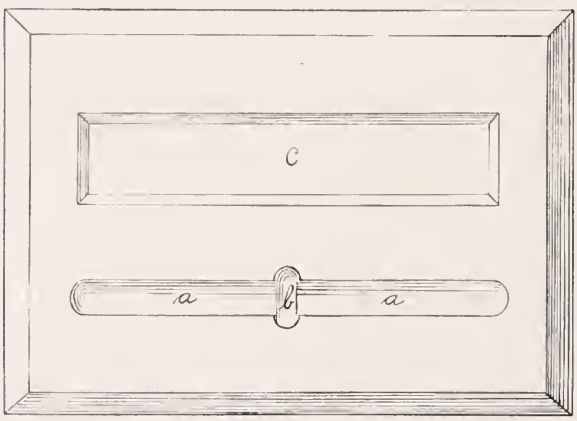

FIG. 344.

FIG. 343. Operating tray equipped for pulp treatment. Two porcelain trays contain sterile cotton pellets; the larger porcelain tray contains six broach holders and broaches, with sterile cotton wrapped on. These have just been removed from the dry sterilizer. The glass slab for sterilizing broaches is better shown in Figure 344. Two of the small square porcelain trays contain medicaments, the third gutta-percha points.

FIG. 344. Glass slab for sterilizing broaches and other instruments used in pulp treatments. The depression, a, a, is filled with ninety-five per cent phenol; c is filled with alcohol, using the dropper bottles shown in Figures 345, 346. Broaches or burs may be immersed in the phenol, then picked up with the pliers (b is more deeply recessed for this purpose) and washed in alcohol. Broaches may be resterilized as often as desired during the operation.

Fig. 345.

Fig. 346.

Figs. 345, 346. A very convenient dropper bottle designed by the late Dr. J. Austin Dunn. After cap is removed and the bottle tipped forward, as in Figure 346, slight pressure on the bulb forces out a drop at a time.

Fig 347 Beechwood Creosote

Fig. 348. Oil of Cloves

Fig. 349. Oil of Cinnamon

Fig. 350. "1—2—3"

Fig. 351. Cresol and Formalin

Fig. 352 Cresol and Formalin

FIGS. 347 TO 351. Direct color photographic reproductions of portions of forearms upon which various antiseptics were sealed under rubber covers for forty-eight hours, to determine their effect upon the tissues. In each instance a small pellet of cotton was moistened with three drops of the medicament and this was sealed on under a piece of rubber dam, as shown in Figure 353. The photographs were taken immediately after the removal of the applications, which were placed on the arms of members of the class of 1915, Northwestern University Dental School.

FIG. 347. Beechwood creosote. There was no discomfort, and when the drug was removed the skin appeared to be very slightly stained.

FIG. 348. Oil of cloves. There was no discomfort, and within two minutes after the application was removed the spot could not be seen. There is some discoloration of the portion of the arm in the lower part of the illustration; this was caused by the adhesive strip.

FIG. 349. Oil of cinnamon. There was marked inflammation. A large blister formed and burst within thirty-six hours.

FIG. 350. "1-2-3." This caused a slight irritation and there was one little blister when it was removed.

FIGS. 351, 352. Cresol and formalin. All of the arms to which this drug was applied were painful; one student removed the application after seven hours. These two fairly represent the inflammation resulting from a forty-eight-hour application. These centers were yellow and looked as though the tissue would slough off, but it did not. Two months later, induration was still present and the dead tissue was being gradually thrown off in scab after scab.

apical end of the canal; and when the broach is withdrawn, the cotton will remain in the canal.

Surgically clean fingers. Even with the equipment above, it will occasionally be necessary at the time of operating to use the fingers in wrapping a broach. For this very small quantities of cotton are placed in little envelopes and sterilized in the oven. One of these may be stuck into a slot in the operating tray shown in Figure 343, and the end cut off with the scissors, so that the cotton may be reached either with the pliers or fingers. The hands should be thoroughly scrubbed and then immersed in an antiseptic solution before the cotton or other aseptic material is touched.

The carrying out of these simple plans of asepsis presents no difficulties nor delays, if the necessary equipment is at hand and conveniently arranged.

Sealing treatments.

The material used for sealing treatments should be absolutely impervious to penetration by the fluids of the mouth; it should hermetically seal the cavity; it should be easy of manipulation; it should be sufficiently hard to withstand the stress of mastication without very much wear; it should be easily removable from the cavity when desired. Pure base-plate gutta-percha is the only material I know which meets these requirements. The so-called temporary stoppings contain so much wax that they are too soft to be dependable. Careful experiments conducted in my laboratory by Dr. George C. Poundstone* have shown that the oxyphosphate cements can not be generally relied upon as being impervious to moisture. This is also shown by the number of teeth which discolor when cement is used as a sealing agent for a considerable time. Another objection to cement is the difficulty of removing it. If the cement has become thoroughly hard, it often requires much cutting with a bur. This is usually an unpleasant procedure for the patient, and may be very painful if the tooth is tender to pressure.

Technic of sealing treatments with base-plate gutta-percha. The technic of placing a filling of base-plate gutta-percha is simple, but the rules must be followed very exactly to insure success. It is essential in the first place that the cavity

* Microscopic Study of Cements, Transactions Illinois State Dental Society, 1904, p. 82; Dental Digest, Vol. XI, 1904, p. 1.

Cements, Transactions Illinois State Dental Society, 1905, p. 136; Dental Review, Vol. 19, 1905, p. 802.

Cement Problem in Inlay Work, read before Fourth International Dental Congress, 1904, Dental Cosmos, Vol. 47, 1905, p. 756.

should be cut to reasonably good form for the retention of the gutta-percha filling. The pulp being involved, the cavity is necessarily of some depth and it will usually require very little additional cutting to give sufficiently good retention form.

The rubber dam should be on and the cavity thoroughly dry. The walls should then be slightly moistened with a eucalyptol* which will dissolve gutta-percha. This will soften the gutta-percha which comes in contact with the walls so that it will adhere. The gutta-percha should be warmed until it is quite soft and pliable. Care should be taken not to overheat it, as it will not again become as hard as it otherwise would. If the cavity is small, the filling may be made of a single piece, but for most cavities it will be best to use several small pieces, placing each one and packing it carefully with as large a flat-end instrument as can be used in the cavity. I prefer to use amalgam pluggers, which have flat, serrated ends. The direction of the force should be toward the walls, the same as in packing gold. Additional pieces should be added until the cavity is full. All of the packing should be done with cold instruments. Then with a flat burnisher, heated sufficiently that it will readily cut the gutta-percha, the filling should be trimmed to form. The movements of the hot burnisher should generally be toward the margins. If the burnisher is not heated sufficiently, it will drag in the gutta-percha and loosen it. It must be hot enough so that it may be carried through the gutta-percha with a quick stroke. When the trimming is done in this way, the mass of gutta-percha will not be heated sufficiently to cause pain.

In proximo-occlusal cavities, the septal tissue should be protected by holding the blade of a finishing-knife, or other suitable instrument against the tooth at the position of the gingival wall. This will prevent the gutta-percha from being crowded against the soft tissue.

If, in the sealing of a treatment with gutta-percha, it is desired to avoid pressure, a piece of stiff writing-paper may be laid in the cavity and covered with a layer of cement, or a piece of sheet metal — copper, German silver or steel — may be placed first, and the gutta-percha sealing placed over it. When

* There are some thirty-odd species of eucalyptus trees, each of which furnishes an oil, but these oils differ very widely. Some will dissolve gutta-percha very readily; others will not dissolve it at all. Only those which dissolve gutta-percha readily should be used for this purpose. The oil may be tested by dipping a gutta-percha cone into it and then rolling the cone between the fingers. If the oil dissolves the gutta-percha readily, the fingers will be smeared with gutta-percha, and this will adhere very tenaciously to the skin. If the oil does not dissolve the gutta-percha, it should be discarded and search made for an oil which will.

FIGS. 353 TO 358. Reproductions of photographs of portions of forearms upon which antiseptics were sealed under rubber covers for forty-eight hours, to determine their effect upon the tissues. Applications were applied to arms of members of the class of 1915, Northwestern University Dental School.

FIG. 353. Method of applying. A pellet of cotton was moistened with three drops of the drug and applied directly to the arm. This was covered by a piece of rubber dam and sealed with adhesive plaster. It was removed after forty-eight hours.

FIG. 354. Oil of cloves. There was no appreciable inflammation.

FIG. 355. Oil of cinnamon. This is the same arm as shown in Figure 349. A large blister formed and the surface tissue was destroyed.

FIG. 356. Oil of cassia. The area was a brownish red. Neither the pain nor the inflammation were as severe as reported by Dr. Peck in 1898, doubtless due to the difference in the purity of the oils. In fact, the oil used in this experiment was probably not cassia, although it was claimed to be a cassia oil from China.

FIGS. 357, 358. Cresol and formalin. The photographs of these two arms were taken seven days after the application was removed. Figure 358 shows three pins which were pushed in 8 mm. (nearly ⅓ of an inch) before any sensation was felt. This arm looked practically the same two months later.

Fig. 353. Application.

Fig. 354. Oil of cloves.

Fig. 355. Oil of cinnamon.

Fig. 356. Oil of cassia.

Fig. 357. Cresol and formalin.

Fig. 358. Cresol and formalin.

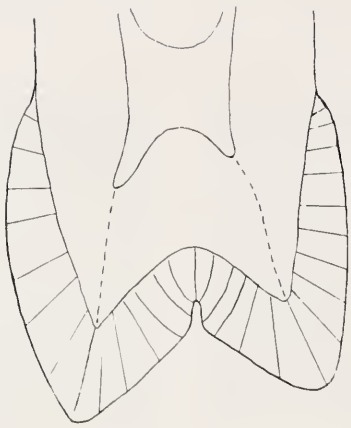

Fig. 359.

Fig. 359. Diagram of a bicuspid tooth split bucco-lingually, showing the directions of the enamel rods in the different parts of the plane of the cut. The recessional lines of the horns of the pulp are shown by the dotted lines.

FIG. 360.

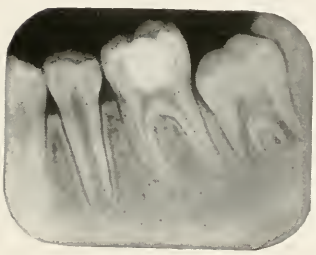

FIG. 361.

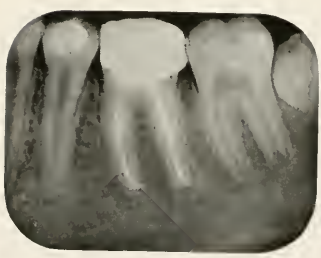

FIG. 362.

FIG. 360. Radiograph of upper incisors, boy eight years old. The large openings in the ends of the root canals are shown.

FIG. 361. Radiograph showing partially developed roots of second bicuspid and second molar at twelve years.

FIG. 362. Radiograph showing partially developed roots of second molar at fifteen years; also a very good root filling in the first molar.

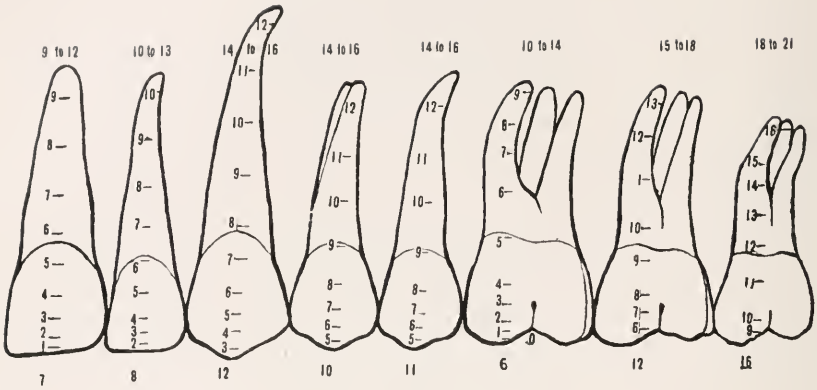

Fig. 363.

Fig. 363. A diagrammatic representation of the calcification of the permanent teeth. The teeth of the left side of the upper jaw are represented in outline. Below each tooth a figure is placed which represents the average year of the eruption of that tooth. Upon each tooth figures are placed at intervals representing the date, in years, of the progress of its calcification to that time. The relation of the progress of calcification between the different teeth, or the contemporaneous calcification lines, may be found by following any individual figure from tooth to tooth. The figure 7, for instance, is at the junction of the middle and gingival thirds of the root of the central incisor and, reading from left to right, it gradually drops down to a little below midlength of the crown of the second bicuspid; it then jumps to about half length of the root of the first molar; then back to the junction of the occlusal and middle thirds of the crown of the second molar; it does not appear at all on the third molar. Any other year may be followed in the same way. The first of the two figures placed above each tooth represents a date at which the apex of the root of that tooth has frequently been found sufficiently narrowed to permit of root filling. The second figure represents the date at which the apex of the root is occasionally found too widely open for root filling. Even wider variations will sometimes be found. It must be remembered that in such a diagrammatic representation, only an approximation to a general average can be expected. Tolerably wide variations will occur.

desired for appearance, a gutta-percha sealing may be placed within the cavity, without entirely filling it, and it may then be covered with cement or temporary stopping.

Rationale of pulp and root canal medication.

Aside from the measures which may be taken to prevent diseases of the pulp, there is little that may be done in treatment which does not involve the removal of the pulp and the filling of the root canals. Therefore, except in cases of hyperemia, treatment is generally not undertaken with the idea of saving the pulp. In operations for pulp removal and root filling, we should have several things prominently in mind: (1) To avoid pain as much as is possible; (2) to maintain strict asepsis; (3) to avoid injury to the tissues about the apex of the root either by infection through the root canal or by the medicines used.

If the pulp is vital, the patient may present complaining of pain. This may be caused by pressure within the pulp chamber; or by an inflammation of the pulp tissue without pressure, the pulp chamber being exposed by a cavity in the tooth. In the former case the opening of the pulp chamber will relieve the congestion and reduce the pain, and the operations for the destruction and removal of the pulp may be undertaken at once. If the pain is caused principally by inflammation of the pulp, a medicament is indicated to reduce the inflammation, and in my hands nothing has proven more satisfactory than oil of cloves or the "1-2-3"* preparation for this purpose. It is also important to protect such a pulp from thermal shock and it should at the same time be protected against infection. Therefore, the medicament should be sealed in, and should usually remain for a week.

When a pulp has been removed, the indication is for a medicament which will keep the root canal sterile. For this purpose any mild antiseptic may be used. In sealing medicaments in root canals, it should always be kept in mind that the drug may penetrate the apical foramen and come in contact with the tissues about the apex of the root. The rule should be that no drug which would seriously injure the soft tissues, if held in contact with them, should be sealed in a root canal. Therefore, such drugs as 95 per cent phenol, oil of cassia, or preparations con-

* One part oil of cassia, two parts of phenol, three parts oil of gaultheria. The oils should be mixed, and melted crystals of phenol should be added. This makes a clear solution. If 95 per cent phenol be added, it will make a cloudy solution. I have not personally been responsible for the term "1-2-3," which has become a common term, but I have accepted it and mentioned it often in what I have written.

taining formalin which will injure the tissues, should never be sealed in root canals. Certainly no drug should be sealed in a root canal, which would cause serious injury to soft tissue elsewhere, as the skin, when held in contact with it. The use of such drugs has, doubtless, so injured the apical tissues as to lead to the formation of a fair percentage of incurable chronic alveolar abscesses.

Experiments with medicaments used in pulp treatment. During the winter of 1897-98, Dr. A. H. Peck, who was at the time associated with me as a teacher in Northwestern University Dental School, conducted an extended series of experiments to determine both the antiseptic and irritating properties of various drugs used in root canal medication. Among other things, Dr. Peck took pellets of cotton saturated with these drugs and sealed them to the skin of guinea pigs, and to the skin of his own person as well, under rubber cups, for varying lengths of time. These experiments, which were reported to the Illinois State Dental Society,* proved very conclusively that many of the drugs then in use were not only poisonous to the vegetable cell, but to animal cells as well. Such drugs are not indicated in root canals.

I quote part of Dr. Peck's report on oil of cassia, and make brief reference to his statements regarding several other drugs:

"As a test of the irritating properties of oil of cassia, a pellet of cotton was saturated with it and placed in a small rubber cup, to prevent evaporation. This was applied to the surface of the skin and held there by means of a piece of court-plaster large enough to cover it over and stick tightly to the surface of the skin about the edges. This was retained in place for twenty-four hours, during which time the irritation to the soft parts was by no means a pleasant feature. At the end of this period a blister invariably forms; however, the inflammation in the tissues at this time is not very great. The blister will occupy an area from one-half to one-third greater than that to which the oil is directly applied, and will fill and refill with serum several times before any tendency to recovery is noticed. At the end of forty-eight hours the inflammation in the parts involved is intense, and occupies an area four or five times as great as that to which the oil is directly applied. Numerous small, independent blisters almost invariably form about the circumference

* The Essential Oils and Some Other Agents, Their Antiseptic Value, also Their Irritating or Nonirritating Properties. By A. H. Peck, Transactions Illinois State Dental Society, 1898, p. 154; Dental Review, Vol. XI, 1898, p. 593.

of the inflamed area. This condition continues for several days, and while the inflammatory process is at its height the sore is one of the ugliest and most formidable in appearance it has ever been my privilege to look upon. These sores, also, are very slow in healing.

"To my mind, it is clearly proven that while the antiseptic and germicidal properties of this oil are of the highest order, it is one of the most irritating, in its effects on the soft tissue, of all the agents with which we have anything to do. And because of these effects, as outlined above, I feel perfectly justified in making the statement that oil of cassia should never be used as a dressing in the root canals of teeth."

Oil of cinnamon, beechwood creosote, oil of cloves, "1-2-3," formalin, and several other medicaments were experimented with in the same way and the results stated. Oil of cinnamon was reported as causing "considerable irritation" but not so much as oil of cassia.* The application of beechwood creosote was "practically nonirritating." Oil of cloves was reported as "absolutely nonirritating. An application to the surface of the skin for thirty-six hours left no more evidence of having been confined there than so much sterilized water would have done." The application of "1-2-3" produced "a slight searing." Formalin was used full strength ("saturated solution of the gas formaldehyde in water") which was at that time recommended for the treatment of root canals. It produced a very severe inflammation, causing marked systemic disturbances, and destroyed a large mass of tissue.

Recently, at my request, Dr. E. S. Willard, professor of bacteriology in Northwestern University Dental School, made a similar series of experiments, sealing three drops of each medicament for twenty-four hours on the forearms of members of the senior class, who volunteered for the purpose. Direct color photographs of six of the arms are reproduced in Figures 347 to 352, and six other photographs are reproduced in black and white in Figures 353 to 358. While other applications were made, those illustrated were selected as representing typical results. Descriptions of the various cases are given in connection with the illustrations. It is therefore only necessary to add a general statement here.

* During the Boxer war in China it was impossible to get oil of cassia, and substitutes were placed on the market. I have been unable to secure pure oil of cassia since. A special effort to do so was made recently, without success.

The most important difference between the results of these experiments and those made by Dr. Peck seventeen years ago is in the inflammation caused by oil of cassia. In the recent tests, it seems to have caused less inflammation, yet far too much to permit of its use in a root canal. The difference is doubtless due to the difference in the purity of the two specimens. Oil of cloves and beechwood creosote each produced practically no inflammation, "1-2-3" only a slight irritation, and oil of cinnamon produced a large blister, exactly duplicating Dr. Peck's results. Cresol and formalin in each instance in Dr. Willard's experiments produced a very deep inflammation which was painful, so much so that one student removed the application after seven hours. This arm showed a scar six weeks later which looked as though the area had been burned. No blister occurred on any of the arms to which this medicament was applied; the tissue turned a yellowish color as though it would slough away. Figure 358 shows one arm photographed on the seventh day after the removal of the drug. Three pins are shown sticking into the arm. One of these was pushed directly in 8 mm. (about 1-3 of an inch) before any sensation was felt. Two months after the application, induration was yet present and the area still looked as though some tissue would be lost. A drug so injurious to tissue should not be sealed in a root canal.

The subject of the use of antiseptics in contact with the tissues, in the irrigation of wounds, is discussed under a separate heading. I have referred to Dr. Peck's paper and reported the recent experiments to impress the fact that those antiseptics which are least irritating to the soft tissues are indicated for sealing in root canals, to avoid the danger of serious injury to the periapical tissues and the establishment of chronic alveolar abscess. The principal purpose of placing a medicament in the canal is to certainly keep the canal sterile, to prevent hematogenous infection of any portions of the pulp which might remain for the time.

Preventive treatment of hyperemia and inflammation of the pulp.

As hyperemia may terminate in inflammation, the preventive treatment should be the same for both. Such treatment will also be preventive in the highest degree against alveolar abscess, and its sequelæ.

In reviewing the various causes of hyperemia of the pulp which have been mentioned, it will be recognized that many cases

may be prevented by greater care in dental operations. In the preparation of cavities care should be taken to avoid near approach to the pulp, or in cavities which are necessarily deep, nonconductors should be placed to avoid the danger of a hyperemia. In those cavities in which two or more walls are even fairly close to the pulp, as in mesio-disto-occlusal cavities, it is well to use a nonconductor under the filling. Every precaution should be taken to avoid the creation of heat or undue pulp irritation in all operating. Attention has already been called to the danger in the use of disks, stones, etc.

THOROUGH AND FREQUENT EXAMINATIONS. Involvement of the pulp by caries may be prevented in proportion as we find and fill cavities before they become deep. We should, then, be very thorough in examinations of the mouth, to find cavities while they are small. We should request patients to come in at stated intervals for examination, in order that cavities may be discovered before the dentin has been deeply penetrated. Patients should be brought to realize the advantage of such a plan.

AVOID NEAR APPROACH AND EXPOSURE OF PULP IN CAVITY PREPARATION. In cavity preparation, the utmost care should be taken, not only to avoid exposing the pulp, but to avoid near approach to the pulp chamber, or to the recessional lines of the pulpal horns. The dentin grows from the dento-enamel junction inward, and the pulp recedes and becomes smaller as the dentin is formed. The lines along which the horns of the pulp recede as the dentin is gradually formed are called the *recessional lines of the pulpal horns.* Oftentimes a horn of a pulp will persist as a slender thread of pulp tissue reaching far toward the dento-enamel junction, even when the pulp chamber has become quite small. A very large percentage of the accidental exposures of the pulp in cavity preparation are exposures of the horns of the pulp. Therefore, it is especially important that these lines be avoided in preparing cavities. In the bicuspids and molars, there is a recessional line for each cusp. This line is in the direction of a line drawn from the point of each cusp toward the corresponding angle or horn of the pulp chamber. (See Figure 359.)

A pulp is in danger of death from thermal shock subsequent to the placing of a filling, if the cavity be cut deeply enough to closely approach the pulp chamber or horns of the pulp at any single point, or if much of the cavity be cut only fairly deep. In other words, a pulp might die from thermal shock from a

metal filling placed in a small deep cavity, or from a very broad, but comparatively shallow cavity. There would be much more danger of thermal shock from a mesio-disto-occlusal filling of moderate depth than from a simple occlusal filling of the same depth, because the mesio-disto-occlusal filling would be in contact with the dentinal tubules on three sides of the pulp and sudden changes of temperature would produce greater shock. A careful review of the cavity forms in my work on Operative Dentistry will show that they are planned to give the best possible resistance and retention forms, and at the same time avoid near approach to the pulp chamber and the recessional lines of pulpal horns. Naturally, no cavity should be cut deeper in the dentin than is required for retention.

Use of nonconductors. In every case in which it is necessary, either in the removal of caries, or in securing proper cavity form, to cut deeply enough to endanger the life of the pulp from thermal shock, a nonconductor should be placed beneath the filling. The technic of placing nonconductors is very simple. In some cases, particularly to cover axial walls of incisor proximal cavities, a piece of quill, cut from a quill toothpick, may be used. This is cut to lie against the axial wall and is held in place until one or two pieces of gold have been condensed, overlapping a margin of the quill. A thin layer of oxyphosphate of zinc cement may be generally used as a nonconductor. This may be conveniently placed by cutting a piece of stiff writing-paper to fit the cavity, and after putting the necessary amount of cement on this, it should be carried to the cavity with the cement next to the dentin wall to be covered, and gentle pressure made on the paper until the cement is spread out into a thin layer.

Treatment of hyperemia.

In cases in which there is evidence of hyperemia which has developed as a result of excessive changes of temperature, the patient should be urged to use the greatest care to avoid hot or cold drinks, hot or cold food, or breathing cold air through the mouth. If these are rigorously avoided for a few days, the milder hyperemias will disappear as the rule. If the patient is one who must be out of doors in cold weather, the sensitive teeth may be protected by a covering of gutta-percha or modeling compound, molded to fit closely, but which may be removed and replaced by the patient at meal time. Some patients will make use of such an appliance, others will be so much irritated by it,

that they will not keep it in the mouth. In some cases the exposed portion of the tooth may be partially or entirely covered with cement. If it happens to be a molar tooth, copper cement may be used, and it should be placed on the occlusal surfaces of the other molars of the same arch on both sides to relieve the occlusion on the hyperemic tooth. If these precautions are not taken, the condition is likely to grow progressively worse. The paroxysms of pain will become more frequent, they will be excited by a less degree of temperature change, and the duration of the paroxysms will gradually increase. Finally the pulp will die.

I have seen cases in which the tooth was so sensitive to temperature changes that water three degrees off the temperature of the body, either too hot or too cold, would excite a severe paroxysm of pain. Even these cases will get well, as the rule, if the utmost care is exercised to avoid temperature changes which produce pain. If paroxysms can be avoided by keeping the tooth at even temperature of the body, there is a good opportunity for recovery.

The difficulty in the treatment of hyperemia is in the control of the patient, or in bringing the patient to a realization of the cause of the condition and of the treatment necessary to allow the pulp to recover. We have some peculiar examples of this. Many patients with hyperemic teeth have told me that the paroxysms of pain were never produced by hot or cold taken into the mouth, yet a dash of cold water or a bit of warm gutta-percha applied to the tooth would produce a paroxysm of pain. Some patients can not be induced to take any care whatever to prevent thermal shock until the pulp has an opportunity to recover.

When a patient presents with a hyperemic tooth, it is a good rule for the dentist to emphasize the fact that one of two things will probably occur; either the pulp will very gradually recover, or the paroxysms may become worse and then the tooth may rather suddenly be entirely free from pain. The patient should be warned that if the latter occurs, the probability is that the pulp has died, and there is the danger that an abscess will develop unless the dead pulp is promptly removed. In any event the dentist should, if possible, have the patient return at stated intervals, in order that he may have definite knowledge of the progress of the case.

Capping exposures of the dental pulp.

In discussing the healing powers of the dental pulp, I gave a historical review of the efforts which have been made to save

exposed pulps by capping, in which it was pointed out that most such attempts resulted disastrously, and the operation should not be undertaken except under the most favorable conditions.

In cases in which there occurs in the preparation of a cavity slight exposure of a previously uninflamed pulp in the mouth of a young person, the effort should often be made to save such a pulp by capping. This is especially important if the age of the patient is such that there might be some question as to complete calcification of the root. While, as a rule, it would be expected that the pulp would die, there remains the possibility that it may live to complete the formation of the root. Whenever a capping is undertaken under these conditions, a temporary filling should be placed in the cavity and the vitality of the pulp tested at stated intervals, so that the pulp may be promptly removed if it dies. If, upon the removal of the pulp, it should be found that the apical foramen is so large that it is impossible to make a proper root filling, the only alternative is to extract the tooth.

It became my habit of practice at quite an early date, to make a capping in cases occurring in the teeth of children where the prospect seemed favorable and, if this failed, either to remove the pulps or to extract the teeth. I should advise strongly that in such cases we should abandon the case at once if the first capping, which seemed to have been judiciously made, fails. Every renewed attack of pain marks an extension of the inflammation in such cases. The first effort has, therefore, been made under the very best conditions which could occur in the particular case. A repetition of the effort is simply to worry the child without accomplishing anything in such a large proportion of the cases that it is not justifiable.

The operation of capping should be generally employed in cases in which a slight exposure has occurred during the childhood period of the permanent teeth,* even though this exposure has been made by caries. During this period the apical end of the partially formed root is wide open, so that there is little danger of the death of the pulp from strangulation. There is room for both arteries and veins to become enlarged. (See Figures 360, 361 and 362.) Such pulps, if exposed by caries, will generally die; although a few live. If slightly exposed in excavating, a considerable number will live if carefully capped and protected from further irritation.

* For each tooth, the period between the time of its eruption and the complete calcification of the root is the childhood period. (See Fig. 363.)

Time of complete calcification of the roots of the various teeth. I reproduce herewith an illustration from my work on Operative Dentistry, showing the average time of the sufficient narrowing of the apical foramen for root filling in the various teeth, with something of the variations which occur in this process. (See Figure 363.) Cases will be found, however, in which the roots of teeth should not be filled so early as the ages mentioned, and this should always be determined by examination of the individual case. It is the habit of many dentists to attempt to fill the roots of teeth of children at too early an age, and hence disastrous results occur.

The examinations for determining the time of the narrowing of the apical foramen sufficiently for root filling, have been carried out by actual measurements in cases in which I have prepared to fill the roots and have found the apical foramen still too broad, or even funnel-shaped, necessitating the extraction of the teeth; also by the examination of the width of the apical foramen in a large number of teeth extracted for children, noting their ages in both these conditions, and making records of them; also by radiographs taken especially for this purpose. Dr. C. F. B. Stowell reported in the Northwestern Dental Journal, Vol. VIII, p. 57, a tabulation of measurements of the foramina of 4378 teeth extracted during root development. The measurements are practically the same as my own.

Indications for capping. The indications for capping may be summed up as follows: (1) During the childhood period (previous to the time of complete formation of root) while apical foramen is large, whether exposure is by caries or in excavating; (2) slight exposures with hand excavator in fully formed teeth; (3) never in fully formed teeth if exposed by caries; (4) never if exposure is made by a bur.

Technic of capping. If the effort is to be made to save an exposed pulp, the aim should be to avoid as much as possible any further irritation. If there be any hemorrhage, the blood should be absorbed with cotton. The immediate area should then be slightly moistened with a very mild antiseptic, such as oil of cloves or "1-2-3." A strong antiseptic should not be used, on account of the danger of increasing the inflammation of the pulp. The dentin about the exposure should be dried with cotton. Then, if the shape of the cavity will permit, a very thin wafer of pink base-plate gutta-percha, slightly moistened with eucalyptol, should be placed over the exposure. The eucalyptol will render the gutta-percha sufficiently sticky, so that it will

adhere to the dentin. Then a small piece of stiff writing-paper should be cut so that it may be laid in the cavity over the gutta-percha. When this is ready, a mixture of oxyphosphate of zinc cement should be made and a small globule placed on the piece of paper. The paper should then be carried to the cavity and placed with the cement side over the gutta-percha, making gentle pressure on the paper to flatten the cement into a thin layer. If the cavity is too small to permit the use of the wafer of gutta-percha, this may be omitted and the capping made by placing the cement directly over the exposure, using very gentle pressure to avoid forcing the cement into the pulp chamber.

After the cement is thoroughly hard, a temporary filling should be placed, leaving the paper over the cement capping in order that there will be no danger of removing the capping when the temporary filling is removed. In all such cases, tests should be made at frequent intervals to know the condition of the pulp. Permanent fillings should usually be postponed until the formation of the root is certainly completed, or for six months or more in cases in which cappings have been made in adult teeth.

Treatment of Vital Dental Pulps.

Exposure of the dental pulp.

Conditions presenting. The pulp of a tooth (1) may be found exposed by caries so that it lies naked and in view; (2) it may have been reached by the extension of caries but remain covered by a softened carious mass of dentin; (3) it may become exposed by accident during the preparation of a carious cavity.

The first and second cases are so similar that they may be considered together, only noticing differences of manipulation as they occur. In both, the supposition is that the pulp is to be destroyed and removed. In the first procedure, the problem is the preparation of the cavity for the treatment of the exposed pulp, with the least pain and inconvenience to the patient.

Opening the cavity. The requirement is that the cavity be opened by the removal of all overhanging enamel and that the surrounding walls be freed from carious material, perfectly cleaned to solid dentin and cut to a form that will certainly retain a temporary filling for the purpose of sealing in applications that may be required in the treatment. It is not required here that the cavity be cut to the full outline form, as it will be prepared to receive the permanent filling later, nor that the permanent anchorages be provided; but it is required that good and

sufficient anchorage be made for a temporary gutta-percha filling against good, clean surrounding walls in every part. The cavity should be opened sufficiently wide to admit of the free and easy application of instruments for the exposure of the pulp. In doing this, especial care should be taken that the excavators be not directed toward the pulp of the tooth and that it be not interfered with in any way until after the surrounding walls are clean and solid. This excavation should be done upon the principles laid down for the excavation of cavities in the class to which the case in hand belongs.

Rubber dam on. It must be understood that the pulp is not to be exposed or the pulp chamber entered at any time, either primarily or secondarily, without the protection of the rubber dam. If the rubber dam has not been placed at the beginning, it should be placed after the cavity is well opened, and every preparation should be made for the best possible view of the deeper parts of the cavity, and the field of operation sterilized. The carious material should then be removed from the deeper parts of the cavity, and from about the exposure. In case the exposure is large and the pulp is already laid bare, the excavating need not be very perfectly done at first, the necessity being that applications can be laid directly upon the pulp tissue and perfectly sealed in place by a temporary filling. It must be done, however, before any part of the pulp is removed in order to be sure that no infectious material be carried from the cavity into the root canals.

Make exposure with broad instrument. In case the pulp is covered with carious material only, this should be removed and the tissue of the pulp laid bare. In every case this should be done with the broadest cutting instrument that is applicable to the position, usually with the spoons. One should never undertake to remove softened material from over a pulp with an instrument so small that it is liable to pass through the opening into the pulp chamber, lacerate the pulp tissue, and inflict unnecessary pain. This should be taken as a principle controlling every procedure in this class of cases, and the operator should see to it particularly that the cavity be so opened and prepared that broad points may be used with facility.

In bicuspids and molars. When these preparations have been made, the best direction in which to make a sweeping cut having been determined, a spoon should be placed with its edge under the carious mass close against one of the walls of the

cavity, and with a strong thrust in a curved direction it should be carried across to the other side, cutting at once to the full depth of the softened dentin. If possible, the whole mass should be removed at a single cut, laying the pulp bare. The position of the spoon for making such a cut is shown in Figure 364. This should be carefully planned and firmly executed. If the cut should be through the superficial portions of the pulp, excising a portion of the tissue, it is just as well, for when the hemorrhage has ceased, we are sure of the best condition for the absorption of remedies for destroying it, whether this be done by the application of arsenic or by use of cocain under pressure. In some broad cavities in which it may seem that the carious mass is too broad to be removed at a single cut, one or more preparatory cuts may be made to either side, avoiding the pulp, before making the principal cut for its exposure. An exposure of the pulp made in this way is usually not very painful, and, even if it be very sensitive, the duration of the pain is reduced to the shortest limit of time.

In proximal cavities in incisors. In proximal cavities in the incisors, the spoons 20-9-12* generally can not be used for want of room. Much oftener the spoons 15-8-12 or the discoid are applicable. In these cavities the most desirable direction for the final cut for exposing the pulp is from the gingival toward the incisal, directly over the pulp. In these cases the opening into the pulp is apt to be long gingivo-incisally, and if the broad cutting edge can be placed at right angles to this, it is much safer against dropping into the pulp chamber and producing unnecessary laceration of the sensitive tissues. By proceeding carefully, this position, or an angle closely proximating it, can often be obtained, and then the exposure is made with safety. A discoid is really the best instrument for the purpose in this position. The exposure may be made with spoons 10-6-12, but with more danger of inflicting pain.

Medication to reduce inflammation. If the patient has suffered pain, indicating considerable inflammation of the pulp, it will generally be best, after making the exposure and having produced a slight hemorrhage, to seal in a dressing of oil of cloves for a week to allow time for the inflammation to subside, before proceeding with the destruction of the pulp. If a pulp is very much inflamed, applications of cocain may be extremely painful, without producing anesthesia, and arsenic may cause

* For explanation of instrument formulæ, see Operative Dentistry, Vol. II.

the tooth to ache severely. By delaying, this pain may be avoided.

When pain is uncontrollable. In a limited number of cases, in which the pain is severe and can not be controlled, the only possible procedure will be to remove the pulp at once, or to at least lacerate the pulp tissue sufficiently to produce a free hemorrhage. To do this without an anesthetic will cause excruciating pain for an instant, but relief will follow almost immediately. If it be a molar tooth, a sufficient opening should be made to permit a spoon to be carried into and swept around the pulp chamber; in other teeth, good access having been secured, a broach may be thrust into the canal, withdrawn a little to be sure that it is free, then twisted several times to engage the pulp and remove it.

In these severe cases the area of the tooth may be anesthetized with novocain, injecting the bone about the apex, or injecting the solution into the nerve trunk which supplies the area. After waiting several minutes for the effect of the novocain, the rubber dam may be placed, and the canals thoroughly cleansed.

Destroying the dental pulp with arsenic.

When the pulp has been fully exposed, the cavity should be ready for the application to the pulp without further preparation. If it is to be destroyed by arsenic, a piece of heavy writing-paper or cardboard should be cut of such size and form that it may be easily laid in the cavity to cover the exposure. The walls of the cavity should be moistened with eucalyptol, or oil of cajuput, to prepare them for receiving a gutta-percha filling. Any excess of oil should be removed. A small amount of arsenical paste may be placed upon the piece of paper and applied directly to the exposure, with the arsenical paste turned against the pulp. The paper should be pressed gently to place.

Avoid pressure in sealing. A gutta-percha filling should be placed over the paper, using especial care not to make unnecessary pressure over the exposure of the pulp, as this might cause compression and pain. This gutta-percha filling should be as perfect in its adaptation to the cavity walls as it is possible to make it, in order that there may be no leakage of the arsenic. Unless the cavity is so shallow that there is lack of room, a further protection of the pulp against pressure may be provided by cutting a second piece of paper or cardboard and fitting it over the first. Cement may be mixed rather thin and a globule placed over the paper, and allowed to harden, which will

give opportunity for the use of any reasonable force in making the gutta-percha filling for sealing the cavity. In proximal cavities no overplus of gutta-percha should be allowed to impinge upon the gum septum and cause absorption, as has been mentioned.

Danger of arsenical poisoning. Care should be taken not to use so much of the arsenical paste that it will run around the margins of the paper and be in danger of smearing the walls of the cavity, or possibly coming in contact with the soft tissues about the tooth and destroying them. This is sometimes a serious accident, endangering several teeth by destroying the gum tissue and alveolar process.

After the temporary filling is completed, if there is a possibility that any of the arsenical preparation may have touched the surface of the tooth or adjacent soft tissues, these should be swabbed with cotton saturated with dialized iron, to counteract the injurious effect of the arsenic.

Subsequent treatment if pulp only partly devitalized. Occasionally, after the arsenic has remained in the tooth for forty-eight hours or longer, the pulp will be found only partly devitalized. The bulbous portion may be dead and that in the canals quite sensitive. After removing the bulbous portion, a second application may be made, or if the cavity is sealed with a dressing of oil of cloves for a week, the remainder of the pulp will be found to have died, as a result of the slight amount of arsenic which had been absorbed by it. Or, after the removal of the bulbous portion, the remainder may be anesthetized with cocain and removed at once.

Anesthetizing the dental pulp with cocain.

If it has been decided to anesthetize the pulp with cocain under pressure, the opening into the pulp must be free, and the position such that the after-manipulation can be readily done. The surrounding walls must be sufficient so that the drug may be readily confined under pressure, and the access should be fairly direct. Otherwise arsenic should be used.

When the cavity has been fully prepared — an occlusal cavity in an upper first molar, for example — a sufficient amount of cocain crystals should be dissolved in a drop of sterile water (always made fresh for each case) and a small pellet of cotton saturated with this placed in the cavity upon the exposed pulp. Over this should be placed a pellet of soft, or unvulcanized rubber (used for making vulcanite plates), that will completely fill

the orifice of the cavity and prevent the escape of the solution. Pressure should be made upon this with a broad-faced amalgam plugger. The pressure should be gentle at first and be gradually increased as the pain subsides, watching for evidence of pain in the countenance of the patient, until very heavy pressure can be made. Then, if all has gone well, the vulcanizable rubber and the cotton may be removed and the pulp will be found insensible, and its removal may be proceeded with.

Requires pressure to secure anesthesia. In anesthetizing the pulp in this way, the cavity must be so stopped with the soft rubber as to prevent the escape of the solution along the cavity walls, otherwise, the pressure will fail to force the drug into the pulp tissue and the anesthesia will fail. This is fairly easy of accomplishment in the cavity named above and those of similar form and situation. But in proximal cavities it is often much more difficult to so place the vulcanizable rubber that it will successfully stop the orifice of the cavity. In the molars and bicuspids this may be done by first placing a properly formed piece of rubber against the proximating surface of the adjacent tooth and then folding it over the occlusal portion of the cavity, afterward applying pressure with a broad instrument point selected to fit the cavity to the best advantage. As one becomes expert in this, most cavities may be so handled as to successfully produce anesthesia of the pulp.

When pulp is not actually exposed. In those cases in which the pulp is not actually exposed, so that it is necessary to cut through more or less dentin to expose it, better results will usually be obtained in the use of cocain anesthesia by cutting a small hole in the dentin, about 1 mm. in diameter, with a bibeveled drill, and placing a minute pledget of cotton, saturated with the cocain solution, in this hole. A small piece of vulcanizable rubber should be applied over the hole and pressure made with an instrument that will just fit in the hole. It should be remembered that the effect of the pressure applied will be in inverse proportion to the area of the end of the instrument used, and much better penetration will be obtained with a small instrument if the solution can be confined in a small hole so that it will be forced directly forward and not spread laterally, as it would do if the small instruments were used in a large cavity.

The best penetration of the dentin is obtained in cases in which the cocain solution is forced into normal dentinal tubules. For this reason, it is often an advantage to disregard the cavity

of decay and drill into the dentin and apply the cocain to normal tubules in some other portion of the crown of the tooth. For example, in cases of distal surface cavities in molars, an opening may be made through the occlusal surface, in the area which must be included in the cavity anyhow, and the cocain applied to normal tubules.

Opening the pulp chamber preparatory to removal of the pulp.

When the pulp has been destroyed by arsenic, the first procedure, when the patient has returned for the second sitting, is to adjust the rubber dam. In proximal cavities, in which the gutta-percha filling has been placed firmly against the proximating tooth, the filling should first be cut through with a fine saw, or trimmed away with a sharp finishing-knife, in order to allow the rubber dam to pass. A single, quick cut with a hot flat burnisher will accomplish the same result. When the dam is in position, the field of operation should be sterilized as has been directed. The gutta-percha filling may then be softened by warming a burnisher and passing the end into it and holding it for a moment, when the gutta-percha may be lifted out. The cavity should next be freed from the arsenic paste, if it has been used, and washed out with an antiseptic and dried. The next procedure is the opening of the pulp chamber.

In case the pulp has been anesthetized with cocain, the rubber dam will be in place and ready for the opening of the pulp chamber, so that from this point the procedures in the two cases will be similar. In either case, the pulp should first be pricked cautiously with a very fine broach, to be sure that it has lost its sensibility, for sometimes there is a failure in either way of operating. It is very bad practice to attempt to remove any part of the pulp tissue through a small opening. In the bicuspids and molars, the opening of the pulp chamber consists in the removal of the occlusal portion or dentinal covering and the manner of doing this will depend much upon the extent and location of the decay.

Occlusal cavities in molars. In the occlusal cavities in the molars in which the decay is large, often hoe 6-2-23 can be slipped into the opening, and, using it as a hook, the entire roof of the pulp chamber may be pulled away, uncovering the pulp. But when the dentinal covering is strong, as is usually the case when the opening is only the exposure of one of the horns of the pulp, the better way is to enlarge the opening with a fissure bur. This

should be passed into the pulp chamber through the orifice of the exposure, and when the operator is sufficiently sure in his knowledge of the anatomy, he may cut around the pulp chamber parallel with its axial walls and remove the covering in a single piece. Otherwise the opening may be enlarged by carrying the bur laterally toward the central portion of the covering of the chamber and then carrying it around in a circle. Then hoe 6-2-23 may be passed into the opening and its blade turned under the occlusal wall of the pulp chamber, the overhang determined, and the cutting directed, until the whole extent of the chamber is uncovered. No overhang should be left at any point. In this cutting, the greatest care should be taken that the bur be not pressed into the floor of the chamber and its form marred. It is best to prepare a number of small fissure burs especially for this by grinding the ends smooth on a stone, while rapidly rotating in the engine. With these there will be no danger of marring the floor of the pulp chamber. When the whole of the covering has been removed, it is generally best to enlarge somewhat toward the mesio-buccal angle in order to give better access to the mesio-buccal root canal. This may be done most readily and in the best form by a scraping movement with the cleoid excavator.

The case is now ready for the removal of the pulp. Incidentally much of the tissue of the bulb of the pulp, possibly all of it, will have been removed in doing this cutting, but no attempt should be made to remove the pulp from the canals until this cutting is satisfactorily completed and the cavity cleared of all dentin chips and cuttings. If this is neglected, it will often happen that these cuttings will get into the smaller root canals and stop them so that they can not again be opened. For this reason all cutting in opening the pulp chamber, especially in bicuspids and molars in which some of the canals are often very small, should be fully completed before any effort is made to remove the pulp from the canals. When in any case it is found that more cutting for access to some one root canal must be made, a bit of cotton should be placed loosely in the root canals that have been opened, to remain while the cutting is being done and until the cavity is again freed from cuttings. Then with the removal of this cotton the last of the cuttings will be removed.

In many cases, after the first opening has been made, the occlusal wall of the pulp chamber can be cut away more quickly with the chisel and mallet.

Proximal cavities in molars. If the exposure is from a mesial cavity, the cutting will be, of course, to the distal and often will involve the removal of the middle third of the occlusal surface with the whole of the dentin intervening between it and the pulp. If a distal cavity, the middle third bucco-lingually of the occlusal surface, with the intervening dentin, should at once be removed to a point well toward the mesial marginal ridge.

Cavities in bicuspids. In the bicuspids the exposures are almost uniformly from cavities in the proximal surfaces, and the pulp chambers are broad bucco-lingually. The cutting for opening the pulp chamber must be directed first to the central part of the crown, but later broadened from buccal to lingual; for the horns of the pulp when long are inclined toward the points of the cusps, as in Figure 365. These horns should be fully opened so that they may be cleaned and solidly filled. The root canals in these teeth, especially in the upper first bicuspids, are given off from the extreme buccal and extreme lingual portions of the chamber, as shown in Figure 365, and unless the cutting is broad in these directions, the broach will not have direct entrance into them.

Cavities in incisors and cuspids. In the incisors and cuspids, exposures are generally from proximal cavities. In opening these for the removal of the pulp, the orifice of the exposure should be first extended to the gingival wall of the cavity and to the full breadth of the chamber. The approach should be carefully considered. Generally a broach will not readily slide into the canal without being bent more or less. (See Figure 366.) This is unfavorable, and a better approach must be made. When a cavity is so large that the pulp has been reached, the lingual wall should generally be cut away, and this will improve the approach, the instrument being passed to the lingual of the incisal edge of the tooth; rarely the labial wall should be cut away. The approach may be improved still more by taking a small fissure bur in the engine, and, approaching the canal from the direction in which a broach would be introduced, passing it into the canal and cutting by lateral pressure, broaden the canal in a direction to straighten the approach, as shown in Figure 367. This cutting will be toward the disto-lingual if a distal cavity, or mesio-lingual if a mesial cavity, if the approach is to the lingual of the incisal edge. From whatever the direction of the approach, the cutting is to be so directed to the broadening of the incisal portion of the canal that the broach will reach the

apex of the tooth with the least bending. In this cutting, special care should be taken that the end of the bur does not cut the opposite side of the canal and roughen it, for, if it should, the point of the broach may catch in the rough points at every effort to introduce it into the canal. By this cutting the curve of the instruments introduced into the canal for the removal of the pulp, or for filling the canal, will be much less abrupt and these operations can be done more perfectly.

Removal of the pulp.

Broaches. The instruments used for removing the pulp from the canals are the barbed broach and the smooth broach. Generally the barbed broach should be used first. Usually the bulb of the pulp will have been removed during the opening of the pulp chamber, and the broach selected should be suited in size to the canal. Each broach should be tested before using it by placing the end against the bottom of the glass dish with sufficient pressure to bend it so that the point will be at a right angle to the handle, at the same time rotating the broach. It should bend in a regular curve. Occasionally in cutting the barbs, the shaft is cut too deeply at some point, which will cause it to break easily; such a broach should be discarded. The broach should generally be held in a light handle, but may be used without. Just before introducing the broach, it should be sterilized by immersing it in phenol and then washing it in alcohol, as has been described. There should be enough of these medicaments so that the working part of the broach can be effectively washed.

It is not meant that this dipping of a broach in the medicaments mentioned is sufficient sterilization for a broach which has been used previously. I would, however, consider this procedure safe for a new broach, or one which, after having been used, had been properly sterilized and then laid aside.

After a broach has been used it should first be cleaned of all shreds of tissue by the use of a stiff brush. If a wire brush is used for this purpose, the motions in cleaning should be from the point of the broach toward the handle, thus avoiding dulling the sharp barbs. The broach may then be immersed for a time in phenol, or some strong antiseptic, in a dish kept for the purpose. Upon removing the broach from the phenol, it should be washed in alcohol, and put away in a small glass bottle. Several bottles should be used for this purpose, so that the different sizes may be kept sorted.

As a general rule I think it is the most satisfactory and economical plan to use a new broach for the removal of each pulp. Often several new ones should be used in molar teeth. This sharp broach will usually bring away practically the entire pulp at once, while a dull broach may bring only a part, thus increasing the difficulty of removing the remainder, and adding much to the time required for the operation. Used broaches may be employed for removing dressings previously placed, or for pulp removal, depending on the sharpness of the barbs.

Technic of removal. The broach should be passed into the canal, the point being directed against one of the walls so that it will pass in beside the pulp tissue rather than through it. Generally the point should be pushed to the apical foramen and then, if it is felt to be held tightly in the apical end of the canal, withdrawn until it is felt to be loose. The broach should then be rotated lightly, moving it slightly back and forth to be sure that the whole length is rotating and not being held in some curved part of the canal which would be liable to break the broach. The rotation should not exceed one turn. The broach should then be withdrawn. In a good many cases the entire contents of the canal will be brought away with the first effort. If not, the movement should be repeated. Sometimes the tissue of the pulp will break up into shreds and be but partially removed. In such cases, the smooth broach with cotton should be used.

After the removal of the pulp, a mild antiseptic, such as oil of cloves or "1-2-3," should be sealed in the canal. Cotton, properly wrapped on a broach, should be saturated with the desired drug, and the excess removed between the folds of a piece of sterile gauze before being placed in the canal. One end of the cotton wisp should project into the pulp chamber in order that it may readily be removed at another sitting. This should be covered with a pellet of cotton similarly treated and the cavity sealed with gutta-percha.

Location of canals in upper molars. Difficulty often occurs in finding the canals in the molar teeth. The difficulty is generally because the floor of the pulp chamber has been mutilated with burs and the openings of the canals filled with chips.

The floor of the pulp chamber is rounded or arched in the center and falls away toward the mouths of the canals. In upper molars, the canals are situated in the position of the angles of a triangle (the molar triangle), shown in Figures 368 and 369, the mesial line of which is the longest, the buccal the

shortest, and the distal the intermediate length. For the first molar, this triangle is well shown in the illustrations representing sections a little rootwise from the floor of the pulp chamber. This is best seen in the specimen itself; and the position and the direction of the canals, with relation to the walls of the pulp chamber and the main points of the surface of the crown, should be carefully studied.

The opening into the lingual root is the simplest and most direct. Generally, the canal begins in a funnel-shaped opening inclining to the lingual, as in Figure 371, which quickly narrows to the dimensions of a moderately small canal and continues to taper to the apical foramen. It is usually very nearly straight. The approach to the canal with the broach is from the buccal, with a lingual inclination. The broach should be placed against the lingual wall and slid forward until it glides into the canal.

The opening of the mesio-buccal canal is under the mesio-buccal cusp, close against the mesio-buccal angle of the pulp chamber. It often happens that this canal opens in a groove in the angle of the chamber, Figures 368 and 369, making this the thinnest point in the dentinal walls surrounding it. In young teeth, the mouth of the canal is of a flattened funnel shape, which is quickly contracted into a very fine canal; but in the adult it often begins as a fine canal. Its course at first is to the buccal and mesial and then to the distal. It is usually distinctly flattened and often has a thin edge to the lingual. It is often a very difficult canal to clean with a broach. To find this canal, the point of the broach should be directed into the mesio-buccal angle of the pulp chamber, and while held against the wall within this angle, should be slid toward the root. It will rarely fail to glide into the canal.

The disto-buccal canal usually begins abruptly as a fine opening, situated at the disto-buccal angle of the floor of the pulp chamber, Figures 368 and 369, so that a broach pressed into that angle will easily glide into it. But in some instances, especially in the upper second molars, the opening is in the floor of the pulp chamber at a little distance from the immediate angle toward the center of the floor, and then, in positions which limit vision, it is often difficult to find. In teeth much flattened at the neck, the opening of this canal may begin very close to the mouth of the mesial canal, Figure 370, or close against the distal wall, or, anywhere between this point and the disto-buccal angle. The first direction of the canal will vary according to its position. If it is found in a fairly well-defined disto-buccal angle of the

chamber, its direction will be a little inclined to the distal and the broach will penetrate it easily; if in the floor of the chamber, it will sometimes be straight, as in the former case; but, more generally, the first direction will be to the distal and buccal, with considerable curve afterward. If found close to the mesial canal, its course is usually first sharply to the distal, when it swerves rather abruptly toward the apex of root. If found along a smooth or curved distal wall, the course will generally be to the distal and buccal with but little curve. This canal is usually very fine from its beginning, and almost or quite round.

While the canals are similar in all of the upper molars, there are differences in the form of the floor of the pulp chamber that may be briefly generalized. The pulp chamber of the upper second molar, Figure 374, is usually much more flattened mesio-distally than that of the first molar. This changes the relation of the openings of the canals somewhat, rendering the distal angle of the triangle formed by them more obtuse and brings the opening of the distal canal nearer the mesial line of the triangle, so that it seems to be found along the distal wall of the narrowed chamber. In others, it is found in the extreme buccal portion crowded close against the mouth of the mesial canal.

The position of the openings of the canals in the upper third molar, Figures 376 and 377, is usually much the same as in the first and second, varying so as to resemble either. Occasionally there are more than the usual number; occasionally only one or two canals. When there is but one, it is commonly quite large. Four, five, or even seven or eight, are sometimes found.

Location of canals in lower molars. The pulp chambers of the lower molars, Figures 378 to 388, have the same general form as the surface of the crowns, but are generally rather more angular. The wall of the chamber toward the occlusal surface is convex toward the pulp; the horns extend from the extreme angles toward the apex of each cusp. The floor, through the central portion, is arched or convex from mesial to distal, and concave from buccal to lingual. The mesial wall of the cavity is flat and longer than the distal, which is rounded or concave. The mesio-buccal and mesio-lingual angles are sharp and projecting, while the distal angles are rounded, Figure 381. The size of the chamber varies much. In youth, its diameter is often as much as two-fifths of the crown and seldom less than one-third. This diminishes as age advances, and in old age it is often very small; especially where there has been considerable

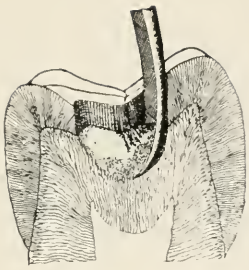

FIG. 364.

FIG. 365.

FIG. 366.

FIG. 367.

FIG. 364. Removing softened material with spoon 20-9-12. In this case this is done before squaring up the dentin walls because there is believed to be danger of exposing the pulp.

FIG. 365. A photograph of a second bicuspid split bucco-lingually to show the form of the pulp chamber. This patient was about fifteen years of age and the illustration shows about the extreme bucco-lingual breadth of the pulp chamber in young persons. In this case the horns of the pulp are rounded, but they are often pointed. This case illustrates the cutting bucco-lingually which is often necessary to fully expose and clean these pulp chambers. Every part of the pulpal horns should be exposed. The dentin was tinged with eosin to sharply distinguish the enamel cap.

FIG. 366. An outline drawing of a central incisor, with an excavated cavity exposing the pulp, split mesio-distally to show the relations to the pulp chamber and canal to the cavity. The canal and chamber are of such breadth as is usually found in young persons. It will be seen that it would be difficult to pass a broach to the apex of the root canal because of the short bend that would be required in entering the pulp chamber.

FIG. 367. A fissure bur is entered into the opening into the pulp chamber and the canal enlarged, as shown, in such direction as to make the use of the broach easy. The canal is then cleaned with much more ease and certainty of the complete removal of the pulp.

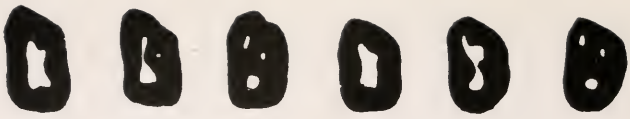

FIG. 368. FIG. 369.

FIG. 370. FIG. 371. FIG. 372. FIG. 373.

FIG. 374. FIG. 375. FIG. 376. FIG. 377.

FIGS. 368, 369. Each of these represents three horizontal sections across the neck and root of an upper first molar. The first in each, reading from left to right, is through the central part of the pulp chamber. The second is at the point where the canals are dividing from the chamber. The third section is a little rootwise from the pulp chamber and shows the molar triangle, formed by the relative position of the canals, to advantage. This exhibits the relation of the root canals to the pulp chamber.

FIGS. 370 TO 373. Lengthwise sections of upper first molars exhibiting the relation of the root canals to the pulp chamber.

FIG. 370. A section exposing the pulp chamber and the canals in the mesio- and disto-buccal roots.

FIG. 371. A section exposing the pulp chamber and mesio-buccal and lingual root canals.

FIG. 372. Another section exposing the two buccal root canals.

FIG. 373. A section exposing the pulp chamber and the canals in the disto-buccal root and the lingual root.

FIG. 374. A perpendicular section of an upper second molar, exposing the pulp chamber and the canals in the mesio- and disto-buccal roots.

FIG. 375. A perpendicular section of an upper second molar, exposing the pulp chamber and the canals in the disto-buccal and lingual roots.

FIGS. 376, 377. The pulp chamber and root canals in upper third molars. Figure 376 is a bucco-lingual section showing the canals in the messio-buccal and lingual roots. Figure 377 is a mesio-distal section showing the divided buccal canals in a tooth with a single root.

FIG. 378. FIG. 379.

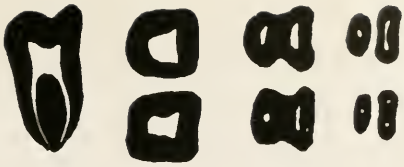

FIG. 380. FIG. 381.

FIG. 382. FIG. 383.

FIG. 384. FIG. 385.

FIG. 386. FIG. 387. FIG. 388.

FIGS. 378, 379. Mesio-distal sections of lower first molars, exposing the pulp chamber and root canals.

FIG. 380. A bucco-lingual section through the mesial root of a lower first molar, showing the pulp chamber and the two canals in the mesial root. Not very infrequently these end in a common apical foramen.

FIG. 381. Six cross sections through the central part of the pulp chambers and the roots of two lower molar teeth. The second cut is just below the pulp chamber in each. The third cut is about midlength of the roots. In the upper series, the mesial root has one broad canal. In the lower series this is divided into two very small canals, widely separated.

FIG. 382. A mesio-distal section of a lower second molar with a single root, showing the pulp chamber and a mesial and a distal root canal.

FIG. 383. A bucco-lingual section of a lower second molar with one root and one large pulp canal.

FIG. 384. A mesio-distal section of a lower second molar with two roots, showing the root canals.

FIG. 385. A bucco-lingual section of the crown and mesial root of a lower second molar, with two root canals which join in the apical third of their length and again separate, ending in separate apical foramina. This is unusual.

FIGS. 386, 387, 388. Mesio-distal sections of lower third molars, showing the forms of the pulp chambers and root canals. Figure 387 shows the single root with one large canal. This is not very uncommon in these teeth.

Fig. 389.

Fig. 389. Photograph showing the position for passing a broach into the canal of the distal root of any one of the lower molars. The removal of the pulp, the cleaning and filling of this particular canal is done best from about this position.

abrasion of the teeth, the pulp chamber may be almost or quite obliterated.

The root canals of the lower molars proceed from the mesial and distal portions of the pulp chamber, Figures 378, 379, 381, 384, 386 and 388. The mesial canal, at its mouth, is usually about as broad from buccal to lingual as the whole breadth of the chamber, including its angular projections. Either at, or a little rootwise from, the floor of the pulp chamber, it is usually divided into two very small canals which diverge at first, and approach each other afterward, but usually remain distinct, each ending in its own apical foramen, Figure 380. Occasionally, however, they are united in the apical third of the root, and end in a common apical foramen. Again, there may be a communication between them in the apical portion of the root, each canal remaining otherwise complete in itself, Figure 385. A few have one broad flattened canal, Figure 381. These canals are usually minute and very difficult to thoroughly clean with the broach, though the mesio-buccal canal is usually easily found if the pulp chamber is thoroughly opened. By placing the point of the broach in the mesio-buccal angle of the chamber and pushing it gently on, it will generally glide into the canal. The first direction inclines to the mesial and buccal, after which it curves to the distal and lingual. Generally, these curves are easy, without short bends. The broach glides into the mesio-lingual canal by placing the point in the mesio-lingual angle of the pulp chamber and sliding it toward the root. The first inclination is to the mesial, but occasionally to the lingual, after which it curves to the distal and buccal.

The distal canal is approached by a funnel-shaped opening, of which the central part of the distal wall of the pulp chamber becomes a portion. Its direction is a little to the distal, and is generally very nearly straight to the apex. At first it is flattened with the long diameter from buccal to lingual, and progressively becomes rounded and tapers regularly to the apical foramen. It is generally much larger than the canals of the mesial root and is easily cleaned with the broach. If the mouth of the patient is wide open and the handle of the broach brought against the upper central incisors with the point directed against the posterior wall of the pulp chamber, it will easily glide into the canal and pass to the apical foramen. This position is shown in the photograph, Figure 389. This particular position for easily entering the distal canal is applicable to all the lower molars. Occasionally, the lower third molar has but one root

canal, Figure 387, which is generally very large. More rarely only a single canal will be found in the lower second molar; but generally, the canals of the second and third lower molars are similar to those of the first. The pulp chambers are usually smaller and oftener irregular in outline. The lower third molar has, occasionally, a very large pulp chamber.

Variations of the forms of pulp chambers. Many variations of form occur in the pulp chambers and root canals. The roots of the teeth may be abnormally crooked. In many instances, the pulp chamber will have in it secondary formations, called nodules, which may be adherent to the walls or block the openings of the canals and prevent a broach from gliding into them. These also occur, occasionally, within the canals, partially blocking the way of the broach. Sometimes the pulp chamber will be filled with nodular deposits so completely that there seems to be no room for the tissues of the pulp. These deposits must be removed before the root canals can be reached and entered, after which the canals will generally be found open. Such deposits occur within the pulp chambers of any of the teeth, but they cause annoyance most frequently in the molars.

Occasionally lateral openings occur from the root canals to the surface of the root. More of these have been seen from the canals of the lower molars than any other teeth. Generally they follow the course of the dentinal tubules and open on the side of the root. They may diverge to one side and curve toward the apex of the root. These can not often be detected, except in dissections of the root, and occur so rarely they may be ignored in practice.

Sometimes the horns of the pulp approach abnormally near the points of the cusps of some of the teeth, as in the upper first bicuspid, and in the mesio-buccal cusp of the upper first molar. Then the pulp is more liable to exposure in excavating carious cavities.

Opening pulp chambers in sound teeth.

Frequently it is necessary to open the pulp chambers of teeth that are sound, or that have fillings previously inserted, the removal of which is not indicated. The pulp may be dead or in such a condition of disease that it should be removed. In these cases, it becomes necessary to cut from the surface of the tooth or through the filling.

In incisors and cuspids. In case of the incisors or cuspids, the best place to enter the pulp chamber is through the central

portion of the lingual surface. For this purpose, a bibeveled drill, one millimeter in diameter, should be first used. Its cutting edges should be very sharp. With this the enamel should be penetrated and the drill forced a little distance into the dentin. This opening should be considerably enlarged by a larger drill or a round bur. Then the small drill should be forced ahead and by several changes of these instruments the pulp chamber may be reached. The point of the small drill should never penetrate very deeply in the small hole. Neglect of this precaution is liable to cause unnecessary pain, or to break the point of the drill by some quick movement of the patient. If the pulp is alive and sensitive, it should now be destroyed. Afterward the complete opening of the chamber may be proceeded with. If the pulp is dead, the further opening of the chamber may be done at once.

In cutting into the pulp chamber through the lingual surface of incisors, the drill has entered from the lingual at a considerable inclination, as shown in Figure 390, and it is necessary to make the opening as nearly parallel with the length of the pulp canal as practicable. To do this, a fissure bur should be used. Its end should be passed into the pulp chamber and the handpiece brought slowly parallel with the long axis of the tooth, cutting from the incisal wall of the opening first made, as shown in Figure 391; then with the same instrument passed farther in, the lingual wall of the pulp chamber should be cut away, going deeper into the root canal carefully, so as not to mar the labial side, until the form shown in Figure 392 is obtained. This cutting should be sufficient to admit a broach to the full length of the canal, with a very little bending. Unless there is reason for delay, as on account of soreness of the tooth, the incisal end of the pulp chamber, which, as in Figure 392, can not be reached for cleaning or filling, should be opened by cutting away the tissue, as shown in Figure 393. This should always be done before a filling is made. Otherwise a little tissue or debris will be left, which will decompose later and discolor the tooth. It is also necessary that this be opened so that it may be solidly filled. When this has been completed, the cleaning and treatment of the canal can be proceeded with.

Generally, when incisors have proximal fillings that are good, the opening into the pulp should be made from the lingual, as above described, without disturbing the fillings. If, however, there is reason for removing a proximal filling, the pulp chamber should be opened through the cavity.

The different teeth of each class show much variation in form and position, which makes considerable differences in the cutting necessary to so straighten the line of approach that the broach and the root canal plugger will go easily to the apex of the root canal, or canals, without so much bending as to interfere with their effective use. Some are of such form, and the line of approach is such, that this is easily obtained while others are very much more difficult. But in almost every case fairly free working of these instruments can be obtained by judicious cutting, which will not be excessive in lines that will materially injure the strength of the teeth. As the future usefulness of the teeth will depend upon the effectiveness of the treatment of the root canals, one should not be satisfied to undertake this without the best access for these instruments that can be reasonably obtained.

In bicuspids and molars. In bicuspids and molars, the opening should be made through the occlusal surface. In bicuspids the mesial pit should be chosen. In molars it is generally much easier to penetrate the enamel through the pit in the central fossa. In this case, as soon as the dentin has been entered it is best to introduce a small inverted cone bur and cut a slot to the mesial inclining to the buccal, and chip the enamel from its margins. The length of this toward the mesial will depend on the position of the tooth and the inclination of the hand-piece in drilling through the dentin. In this, the object is to gain a position from which the drill can be directed into the pulp chamber centrally or toward its mesial portion. The dentin is thick, and, in passing through it from the central pit, this inclination will often carry the hole considerably to the distal. Therefore, in beginning again with the drill, it should be set sufficiently to the mesial so that it will strike the pulp chamber centrally, or to the mesial of its center, as stated. In drilling through the dentin, a small drill, one millimeter in diameter, should first be made to penetrate a little, and then the hole enlarged, then drilled deeper and enlarged, continuing this exchange of instruments until the dentin has been cut through. It should be recognized that there is always danger that a small drill is liable to clog with its chips and to heat, or that it may be broken and the end remain fast in the hole. Or some sudden movement of the patient may break it. For these reasons, a small drill should not be sunk very deeply into the dentin at any time without having enlarged the opening through which it works. The opening should not be made with a large drill in the first instance, because this requires

too much force. If the pulp is alive and sensitive, it should be destroyed; if dead, the opening may at once be so enlarged as to remove the entire roof of the pulp chamber, and the treatment of the canals proceeded with. Treatment of pulp canals should never be undertaken through a small opening.

In a considerable number of cases it is necessary to open the pulp chambers of bicuspid and molar teeth that have been filled. If the fillings are good, proceed as if the tooth were sound, cutting through the filling, or through the dentin, as the case demands. If there is reason for removing the filling, do so at once, and open the pulp chamber through the cavity.

Treatment of teeth having dead pulps.

Conditions presenting. Teeth containing dead pulps may present in any of the following conditions: (1) The pulp may be dead and not infected, having not been exposed to the fluids of the mouth; (2) the pulp may be dead and infected, without having been exposed to the fluids of the mouth; (3) the pulp may be dead and infected, being exposed to the fluids of the mouth. If the pulp is dead and not infected, the soft tissues about the apex of the root will not be inflamed. If the pulp is infected, there may be no disease of the periapical tissues, or there may be an apical pericementitis, an acute alveolar abscess, a chronic alveolar abscess with a sinus, or a chronic alveolar abscess without a sinus — a blind abscess. These will be discussed later, and the time when the dead pulp should be removed will be considered in each condition.

Cases in which the pulp is dead and not infected are necessarily cases in which the pulp has not been exposed to the fluids of the mouth. This condition occurs most frequently from hyperemia induced by thermal shock, less often by accidental blows, etc. Those cases in which the pulp is dead and infected, without having been exposed to the fluids of the mouth, occur from the same causes, the infection having been brought to the pulp through the circulation. There is an occasional case, closely related to this group, in which the pulp dies without having been exposed to the fluids of the mouth by a cavity of decay, but in which an inflammation of the peridental membrane beginning at the gingival line has destroyed the attachment of this tissue entirely to and around the apex of the root, thus causing the death of the pulp. In the multirooted teeth, the pulp may be cut off and infected at the apex of one root and yet more or less of the pulp tissue will be kept alive for a consid-

erable time by the circulation through the apices of the other roots. Such a pulp may give a definite response to a thermal test.

Technic of treatment. Asepsis. In considering the technic of treating such cases, the first proposition is that the pulp chamber and root canals are infected districts, which are to be made aseptic. There seems to be a sentiment that there is no need of aseptic or antiseptic precautions in approaching these, since they are already infected. This is distinctly wrong. In the great majority of cases, the infections are with the mildly pathogenic micro-organisms. Root canals that open into a cavity of decay may contain saprophitic micro-organisms only, which are incapable of spreading into the living soft tissues by growth and producing disease in that way. Their products of decomposition in the root canals may, however, be very irritating and cause an inflammation when passed through the apical foramen. In any of these cases there is always the danger of introducing more virulent pathogenic micro-organisms during the treatment, unless diligent aseptic, or antiseptic precautions are employed in the approach, as in aseptic cases. There should be no difference in this respect whatever in the two classes of cases.

Instrumentation. The instruments used in cleaning the canals are the barbed broach and the smooth broach with absorbent cotton, the same instruments as used in aseptic cases. The rubber dam must protect the parts, and the field of operation must be sterilized. Then if the canals contain fluid, this should be carefully absorbed away as the first procedure by slight wisps of absorbent cotton wound upon the smooth broach. This should not be done by thrusting in as much cotton as the root canal will hold, for the reason that there will be danger of pushing the fluid beyond the apical foramen and causing unnecessary inflammation of the peridental membrane. Very small amounts of cotton should be used, frequently repeated, and the fluid lightly absorbed into these and drawn away without pressure. When the canal is reasonably dry, a barbed broach that enters the canal loosely should be introduced carefully some little distance and withdrawn. The barbs are so cut that they hold debris or shreds of decomposing tissue on the pull and bring away any such material with which they come in contact. This broaching should be continued, washing the broach repeatedly in the phenol and alcohol until the canal is cleaned to its apex. During this process the broach should be inclined this way and that in its

withdrawal so as to effectively scrape all parts of the walls of the canal with barbs, loosening and removing all adhering particles. Finally, the canal should be flooded with a mild antiseptic, such as "1-2-3" or oil of cloves, and the broaching repeated. The medicament should then be absorbed away. This should be repeated until the canal is judged to be well cleaned. Then it should be dried with frequent introduction of very loosely fitting bits of cotton wound upon the smooth broach and so rotated as to entangle and remove any particles which may possibly have been left.

Special care should be taken that the cotton on the smooth broach shall not be in such quantity to form a piston that will push material from the canal through the apex of the root into the tissues beyond; also that the barbed broach be not used in such a way as to gather shreds of material before it and push some of the contents into the tissue beyond the apex of the root. This pushing of material through the apex of the canal, which is liable to produce inflammation, is the one great danger in cleaning infected root canals. It is to be especially guarded against. With the proper sealing of the cavity, the pulp chamber and root canals are a sealed box, the disinfection of which is easily commanded. There is no reason whatever for the use of irritating germicides in its disinfection.

Seal treatment. When the cleaning has been completed, a wisp of cotton wrapped on a broach should first be saturated with the desired medicament and then the surplus should be absorbed by pressing with sterile gauze or cotton, so that the danger of any of the drug passing through the foramen will be reduced to the minimum. This should be placed loosely in the root canal, or in each root canal, when there are more than one. I have generally preferred beechwood creosote in these cases, as it is a sufficiently strong antiseptic and will not seriously injure the apical tissues, in case any of it should penetrate the foramen.

Danger of periapical infection. As mentioned above, the greatest danger in cleaning infected root canals is that some of the infected material will be pushed through the apex. Some have advocated the removal of only a part of the infected tissue at the first sitting and the placing of a medicament to destroy the micro-organisms present in the remainder, removing it at the second appointment. There is danger of causing an infection of the periapical tissues by either plan. The pulp tissue is

usually more or less decomposed and where the pulp is only partly removed there is danger of forcing some of the remainder through the apical foramen in sealing the cavity. Under either plan, the most careful technic should be employed.

It has been the rule of some practitioners to leave the pulp chamber open for a few days after the removal of a dead pulp. This I consider very bad practice. As has already been mentioned, a much more virulent infection may be introduced by failure to follow the most rigid asepsis. These teeth may be comfortable with such treatment, but it may result in the eventual loss of the tooth from alveolar abscess. If the most careful technic is employed at the first sitting, the number of cases in which inflammation and pain will develop will be very few. However, each patient should be told to report if the tooth should become tender or painful. The rubber dam may then be placed, and the treatment removed and replaced at once under aseptic precautions. This will give the same relief as by leaving the tooth open, and new infection is avoided. The danger of periapical inflammation can not be positively eliminated by any plan of treatment.

Treatment of pulp chambers which have been narrowed by calcific deposits.

When the pulp chamber is filled with secondary deposits, the effort should be directed to the removal of these, preserving the outlines of the pulp chamber. When the pulp chamber is much narrowed by secondary dentin deposited upon its walls, the openings into the canals should be found before any cutting is done, and then the cutting carefully directed to straightening them. In most instances this is done best with the barbed broach. All small tortuous canals should be enlarged and straightened with the barbed broach. To do this, the broach should be passed into the canal as far as possible and withdrawn. The barbs will impinge upon the walls and cut away the dentin from the prominent parts of the crooks and straighten them. This should be repeated again and again, pressing the broach in a direction during its withdrawal that will tend most to straighten the canal. By repetitions of the movement, a canal which can be entered by the smallest broach can soon be enlarged sufficiently for filling. When canals are so small that the smallest barbed broach will not enter, a fine smooth broach,

FIG. 390.

FIG. 391.

FIG. 392.

FIG. 393.

FIGS. 390 TO 393. Outline drawings explaining the opening of the pulp chamber and canals in the incisor teeth when this is done through the lingual surface.

FIG. 390. The opening to the pulp chamber from the lingual surface as first made with the drill.

FIG. 391. The opening as modified by a fissure bur, which is placed in the drill hole and inclined so as to cut to the incisal, straightening the approach to the pulp canal.

FIG. 392. The fissure bur is turned to the deeper portion and a cut made lingually, straightening the approach of the broach to the apical portion of the canal.

FIG. 393. Finally the incisal point or edge of the pulp chamber is made accessible by a fissure bur, or a 12-5-12 hoe, as shown in this figure. This part of the pulp chamber can then be thoroughly cleaned and filled. Generally in the incisor teeth of young persons the drill will strike the pulp some distance from its incisal end, as shown in Figure 390, and any neglect to clean and fill this portion is certain to result in a discoloration of the crown of the tooth sooner or later.

Fig. 394. Fig. 395. Fig. 396.

Figs. 394, 395, 396. Three radiographs of the same upper cuspid with a wire in the canal, to show the difference in the shadow-length of the root, as a result of the direction of the rays and the position of the film. With the length of the wire known, the length of the root can be definitely determined from the shadow-length on the film, no matter what the distortion.

which has been roughened with a file, may be used for the first trimming.*

Generally, partial occlusions of canals are confined to or near their pulpal ends, and when these have been enlarged, the broach will pass to the apex. In elderly people certain canals are often too small for successful cleaning and filling. In cases in which there has been much abrasion, the pulp chambers and the pulpal ends of the root canals are apt to be much narrowed by secondary dentin. This applies to all of the teeth in the mouth — i. e., to any that have from any cause, not been worn away, the same as those which are worn.

Removal of calcifications from root canals. If there are many fusiform calcifications in the root canals they will often interfere seriously with passing a broach to, or nearly to the apical foramen. One, who has had experience, will recognize by the sense of touch that the difficulty is due to this kind of calcification. Then the object should be to force the broach as far as possible alongside of such calcifications, and in withdrawing it, obtain room to thrust the broach still further. Finally, if the approach to the canal has been well opened, the broach will catch the mass in such a way as to withdraw it as a whole, cleaning the canal very effectively. Occasionally, however, the whole mass will have to be broken, by continuous probing, cutting out a little at a time until the canal is cleaned of its contents. This is tedious. Often there is a shoulder left near the opening of the canal. If this is cut away, the entire contents of the canal may come away easily. This is an operation requiring experience and a great deal of patience for the best success.

Removal of previous root canal fillings.

A number of cases present with inflammation of the periapical tissues, in which the root canal has been previously treated and a root filling made. It may be that all of the pulp tissue was not removed, and the portion remaining became infected; or the root may not have been filled to the apex and serum collected in the open space and became infected; or the root filling may have been pushed through beyond the apex, causing an inflammation; or some of the peridental membrane about

* To roughen a broach with a file, one should use a flat file cut only one way — not cross cut. The broach should be laid on a piece of moderately hard wood and the file carried diagonally across it, the broach being permitted to roll under the file, while a single motion is made with heavy pressure. The file cuts will tend to cut barbs on the broach in a spiral form around it. If this is properly done, such a broach will cut the dentin quite readily.

the apex may have been destroyed before the dead pulp was removed; or this tissue may have been injured by medicaments sealed in the canal. There may be an apical pericementitis, or an acute or chronic alveolar abscess. The treatment of these will be considered later. We are concerned now only with the technic of the treatment of the root canal.

A radiograph will usually be of great value in determining the difficulties to be encountered and the proper course of procedure. If it is decided to attempt to remove the previous root canal filling, proper access to the canal, or canals, must be had, the same as though the pulp were to be removed. If the root filling has been reasonably well made it will often be impossible to remove it. Supposing the root filling to have been made of gutta-percha, a first effort should be made by heating a root canal plugger and passing it into the gutta-percha. Some of it can usually be removed in this way, and a number of efforts may remove a fair portion. The gutta-percha may be softened beyond the point reached by the end of the plugger, in which case a barbed broach or a twisted broach may bring away the remainder. If these fail, chloroform may be sealed in the canal for twenty-four hours, or eucalyptol may be sealed in for several days, to soften the gutta-percha. It may then be possible to remove it with a broach. The removal of a root filling will often severely tax the patience of the operator, and the most painstaking and persistent efforts may not be successful. One rule should be followed, viz., no instrument in the engine should be used in the effort to reach the apex of a root, on account of the danger of cutting through the side of the root, an accident which practically always results in the loss of the tooth.

Filling Root Canals.

When it is decided that the conditions are right for filling the root canal, or canals, of a tooth, the rubber dam must be placed and the included region disinfected. Then if a treatment has been in the canal, the gutta-percha filling and the dressing should be removed and a critical examination made as to its condition. One principal point is that the canal should be dry.

Size of foramen and length of canal. The size of the apical foramen should be ascertained by trying several sizes of root canal pluggers in the canal, first sterilizing each in phenol and alcohol. Those that are smaller than the foramen will pass through and will be felt by the patient. By beginning with a large point, and trying smaller points in graded sizes, one will

be found that will just pass through the apical foramen. The length of the root may be ascertained by sticking the point of a smooth broach through a little piece of rubber dam and holding the piece of rubber even with the incisal end, occlusal surface, or any convenient landmark on the tooth, while the broach is passed to the apical foramen and is felt by the patient. When the broach is withdrawn, the distance from the rubber to the point will be the length of the tooth. In large canals a broach with a very small hook on the end may be passed through the foramen and will catch on the end of the root, thus giving the length.

The size of the foramen and the length of the root may be very accurately determined with the X-ray. For this purpose, several sizes of brass wire should be kept on hand, and in each case the largest size which may be passed through the apex should be used. A piece of this wire should be passed into the canal until the patient feels it; the other end should then be bent over the edge of the cavity or on the floor of the pulp chamber, the cavity sealed with gutta-percha and a radiograph made. The radiograph will seldom show the real length of the root, but its actual length may be determined by measuring the wire and making the necessary correction in case the wire has been passed beyond the apex or not quite to it. Figures 394, 395 and 396 are reproductions of three radiographs of the same upper cuspid with a wire in the canal, showing variations in the projection of the root shadow on the film. For example, if the wire is 14 mm. long, and in the radiograph it measures 16 mm. while the root in the radiograph measures 20 mm., the actual length of the root would be 17.5 mm., as its length would be exaggerated in the radiograph in the same proportion as the wire.

Technic for large canals. The canal should be flooded with eucalyptol or oil of cajuput, liberally applied upon a wisp of cotton wrapped upon a broach, and the excess dried out with a fresh wisp of cotton. A gutta-percha cone should be selected and about two or three millimeters should be cut off to use. This piece should be of a size which, from the information gained of the size of the apical end of the canal, will be sufficient to fully fill the opening and not be forced through it. A root canal plugger of proper size, tested by passing it into the canal as far as it will need to go, should have the end warmed in the flame, and while holding the piece of gutta-percha cone in the thumb and finger of the left hand, its point should be brought quickly in contact with the large end of the cone and held a moment or until the gutta-percha has stuck to the end of the instrument. After

the gutta-percha has had time to become cold and hard, it should be immersed in phenol and alcohol to render it sterile, then it should be dipped into eucalyptol, which will soften the surface of the gutta-percha slightly. It should then be conveyed to the root canal and carried carefully and firmly into its apical end. On withdrawing the root plugger, the gutta-percha cone will remain, closing the apical end of the root. This procedure should be followed with other bits of gutta-percha cones, cut from larger parts of the cones as the canal is filled to its larger portion, using larger root canal pluggers. After the first two or three pieces, the subsequent ones may be slightly softened by passing them quickly over a flame and directly into the canal. This should be continued until the canal is full.

Rationale of this procedure. By flooding the root canal with eucalyptol or oil of cajuput, the moisture is effectually removed. The oils have a greater affinity, or attraction, for the dentin than has the moisture and therefore displace it. In practice this is a much better method than drying with hot air or hot instruments. The drying is done much more perfectly and more easily. In this operation the cotton wrapped on the broach should never be in such quantity as to force the oil through the apical foramen. Any such action should be strictly avoided. These oils dissolve gutta-percha slightly, and the little oil remaining serves to stick the gutta-percha firmly to the walls of the canal. By putting in the gutta-percha in small pieces, an opportunity is given to pack every portion of the canal and all of its irregularities full.

In filling root canals that are very large at the apical end, as in young persons, care must be exercised that the first cone selected is not so small that it could be forced through into the apical space.

Technic for small canals. In very small canals, in which there is much doubt of being able to reach the apical end, chloro-percha (gutta-percha dissolved in chloroform) should be pumped into them, filling them as completely as possible, and then a small solid cone forced in. This pumping in of chloro-percha is done by wrapping three or four fibers of cotton firmly on a small broach, dipping this in the chloro-percha and conveying it into the canal and pumping it back and forth, repeating the operation until the canal seems to be well filled. A root canal plugger of suitable size may then be thrust into it and some of the chloro-percha forced from the pulpal end. A suitable gutta-percha cone, previously prepared, may be thrust as far into the

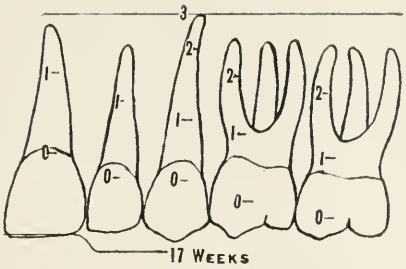

FIG. 397.

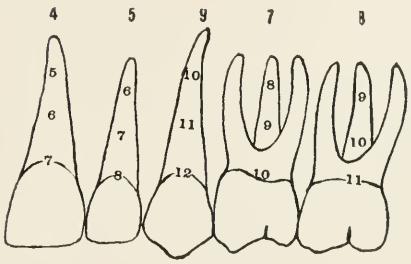

FIG. 398.

FIG. 397. Diagram of the deciduous teeth, considerably enlarged, representing the progress of their calcification. 0, placed upon the individual teeth represents the progress of calcification at birth. The figures 1, 2 and 3 represent, in years, the progress of the calcification of each tooth. The intention is to represent averages. It must be understood that considerable variations will be found.

FIG. 398. Diagram of the deciduous teeth, considerably enlarged, representing the absorption of the roots. The figure placed over each tooth represents, in years, the average time of the beginning of the absorption of its roots. The figures placed upon the roots of the teeth represent, in years, the progress of the absorption of the roots of the several teeth. Considerable variation from the general average, and also in the order of progress, must be expected. Not infrequently the absorption of the root of the second molar is completed before that of the first.

Fig. 399.

Fig. 400.

Fig. 401.

Fig. 399. Radiograph of upper front teeth of boy seven years old. The right deciduous central incisor was abscessed and absorption of the root had not occurred, while the roots of the other three deciduous incisors are being absorbed. The shadow of the root of the right deciduous central incisor may be seen overlapping that of the permanent central crown.

Fig. 400. Radiograph of lower jaw, boy twelve years old, showing deciduous second molar with roots about bicuspid crown.

Fig. 401. Two deciduous molars, which brought the bicuspid germs with them when they were extracted. Quite a few of the missing bicuspids have been lost in this way. Specimens from Northwestern University Dental Museum.

canal as possible. Such canals may not always be perfectly filled by this plan, nor by any other, but in each case the best effort should be made.

In some of the smaller canals, the regular root-canal pluggers will be too large, and a broach of suitable size may be used instead. In the daily use of broaches a large number of these may be collected with which to handle root dressings and for filling canals that are too small for the ordinary root pluggers.

Canals grouped into two classes. While it is impracticable to definitely group the canals of the various teeth into two classes, it may be said that chloro-percha will generally be needed in the upper laterals, first bicuspids and buccal canals of molars, also in the lower incisors and mesial canals of molars. Chloro-percha should generally not be necessary in the upper centrals, cuspids, second bicuspids and lingual canals of molars; nor in the lower cuspids, bicuspids and distal canals of molars. There will be many exceptions owing to variations in the sizes of canals, formations of secondary dentin, the age of the patient, etc.

The pulp chamber should not be filled with gutta-percha. This material is much too soft to serve as a seat for a metallic filling. In any case in which it is not desirable to fill the pulp chamber with the material with which the cavity is to be filled, oxyphosphate of zinc should be used.

To prevent evaporation of chloro-percha. The evaporation of chloroform from chloro-percha may be prevented, in large measure, by keeping the bottle containing the solution upside down. By standing the bottle on its cork, a little of the chloro-percha will pass in between the cork and the opening of the bottle, and as the chloroform evaporates from this portion, the remaining gutta-percha will seal the opening so that no more chloroform may evaporate. The contents of the bottle will therefore remain in a plastic state, ready for use at any time.

Horns of pulp chambers. Attention to the horns of pulp chambers is most urgently demanded in the incisors, cuspids and bicuspids. In incisors particularly, exposure of the pulp, whether made primarily by caries or by cutting into them, are usually at some distance from the incisal end of the pulp, leaving an end protruding into the incisal end of the crown of the tooth. This has been especially noted and illustrated in Figures 390, 391, 392 and 393. It is also true that in cutting into the pulp chambers of bicuspids, one is liable to leave one or both of the horns of the pulp chamber uncleaned and unfilled. Before filling

the cavity, this must be looked for and these openings so exposed that every part of them may be cleaned and filled. Any neglect in this will result in discoloration of the tooth by decomposition of the debris, left in this neglected portion of the pulp chamber. In cuspids and bicuspids, the horns of the pulp are often long and slender and penetrate far toward the ends of the cusps. Unless these are thought of and especially looked for and cut out, so that they may be perfectly filled, discoloration of the tooth in some degree is sure to occur. This may occur in the molars as well, especially in young persons whose teeth have long cusps. Nothing of this kind should escape notice and correction. A study of these points in practice will soon bring such a knowledge of the positions of the horns of the pulp that their exposure will be but a matter of a few strokes of an instrument at the right points.

Treatment of Pulps of the Deciduous Teeth.

There is perhaps nothing that can be considered as more important in the general management of a practice than the proper attention to the deciduous teeth to prevent exposures of pulps in them by caries. These little teeth often begin to decay very early, and constant watchfulness, particularly for decays upon the proximal surfaces, is of paramount importance. If decays expose the pulps, it is difficult for the dentist to handle these teeth successfully afterward.

The exposure may come at a time when it is impossible to make a good root filling. The root may not be fully formed or possibly absorption may have begun, so that the end of the canal can not be properly filled. The period during which the roots of the deciduous teeth are full length is comparatively short, and this is the only time when really good root fillings may be made.

Time of complete calcification and beginning absorption of roots. The accompanying illustrations give about the average progress of calcification of the deciduous teeth, also the average progress of absorption of the roots. There are numerous variations from these. (See Figures 397 and 398.) It will be noticed that the central and lateral incisors are both fully calcified during the second year, and that absorption of the root of the central begins about the fourth year, that of the lateral a year later. The roots of the cuspid and both molars are fully calcified by the end of the third year, while absorption of the root of the cuspid does not begin until the ninth year, the first molar at seven and the second molar at eight. As a general

statement, there is the opportunity to properly fill the root of the central incisor when the child is between the ages of two and four, the root of the lateral incisor between the ages of two and five, of the cuspid between three and nine, the first molar between three and seven, the second molar between three and eight.

Serious results of exposures of pulps of deciduous teeth. It often happens that exposure of the pulp of a deciduous tooth will mean the premature loss of the tooth. This may lead to serious consequences by causing irregularities in the eruption of the permanent teeth. If, for example, the second deciduous molar is lost in this way, there is nothing to prevent the first permanent molar from moving forward so that it laps over the space where the second bicuspid should come through. This condition, or some similar irregularity, is liable to occur as a result of the early extraction of any of the deciduous molars.

One case that came under my observation soon after I began practice has come to my mind many times since. A girl between thirteen and fourteen years old (small for her age), called on me, saying that there was something about her lower jaw that annoyed her, without there being any real pain. I examined the case and noticed that neither lower second bicuspid was in position, and that both first bicuspids were in their proper places, with the first molars inclined very far forward, occupying fully two-thirds of the spaces where the second bicuspids should be. They were one cusp too far mesially in relation to the upper teeth. The lower second deciduous molars had been extracted because of alveolar abscess, according to the history she gave. I passed a sharp, slender instrument through the gum tissue on one side and located the second bicuspid. I also made out that the buccal cusp of the tooth was through the bone.

When she returned, I had determined to restore the space for the second bicuspids, which I did, taking one side at a time, by placing pieces of hickory wood, with the grain in the buccolingual direction, between the first bicuspid and the first molar. The wood was previously compressed in the vise and trimmed to fit accurately. Larger pieces were used as the space was enlarged. Then an appliance was made to hold these teeth apart until the second bicuspid erupted, which it did within a few weeks.

This little girl had had toothache and the pulps had died as a result of neglected decays; she had gone through the ordeal of alveolar abscess and finally the abscessed teeth were extracted. As a result the first permanent molars were tipped forward, and

the second bicuspids were impacted, and if the condition had not been corrected, the malocclusion would probably have led to other difficulties later in life. Many such cases even to-day are passing without correction. If there had been sufficient watchfulness as to decay of these deciduous teeth, the cavities would have been filled early enough to preserve the teeth until the proper time for shedding.

The object of this recital is not to discuss the treatment of such cases, but to impress the fact that such things can be prevented by very simple means if the decays are discovered before the vitality of the pulp is endangered. In this it should be remembered that the pulps of the deciduous teeth are larger in proportion to the size of the teeth than in the permanent teeth, and the wall of dentin is correspondingly thinner, so that a depth of decay that would not be dangerous in a permanent tooth might be decidedly dangerous in a deciduous tooth. Therefore, the watchfulness must be closer and more exacting than in the permanent teeth. Every child should be required to visit the dentist for examination at regular intervals. For most children these examinations should be not more than three months apart.

When pulps of deciduous teeth are exposed by caries, they usually die within a short time. A pulp may die and an alveolar abscess may develop and pass into the chronic form with a sinus discharging through the gum, without much complaint from the child. In many of the cases, however, there is great pain accompanied by fever during the development of an acute alveolar abscess.

Occasionally, deciduous molars will be retained beyond the normal time for shedding. If, for any reason, these teeth are to be extracted, it should be remembered that the germ of the forming bicuspid may be locked between the roots of the deciduous molar, and will be in danger of being extracted with it. (See Figures 400, 401.)

Technic same as for permanent teeth. If a case presents with the pulp exposed, but not yet dead, it may be treated along the same general lines as mentioned for the permanent teeth. Arsenic should be left in the deciduous teeth for a shorter time, usually about twenty-four hours. Oil of cloves may be sealed in for a week, when the pulp will usually be found to be dead, or so nearly so that it may be removed without causing much pain.

The subsequent treatment, including the root filling, should be the same as for permanent teeth, for unless every detail is

properly carried out abscesses are likely to occur. The rubber dam should be in place and every precaution should be taken as to asepsis. In carrying out the treatment of these cases, everything should be prepared in advance so that the shortest possible time will be occupied in the operations. The dentist should have in mind distinctly that he is dealing with a child and often can not keep the child in the chair as long as may be necessary to do all that he might wish at one time. Therefore, he must make provision for cutting short the treatment before it is completed, placing quickly a dressing and a temporary filling, and dismissing the child until a subsequent day.

Chronic abscesses. Chronic abscesses from deciduous teeth will often heal, following proper pulp treatment and root filling. In cases in which these abscesses can not be cured, the teeth should be extracted. It should be remembered that absorption of the root of a deciduous tooth does not occur if there is a chronic abscess, the activity of the absorption cells being prevented by the presence of the suppurative focus. Thus irregularities of the permanent teeth may be caused by the failure of absorption of the roots of abscessed deciduous teeth.

The accompanying illustration, Figure 399, shows one deciduous upper incisor, the root of which has not been absorbed because of an abscess, while the roots of the other three incisors are almost entirely absorbed.

Better care desirable. I have come to feel that the treatment of these cases has not been as careful and painstaking as it should be. Most dentists seem to have carried the teeth along with the least discomfort possible, not succeeding in making their treatment effective. Children are so difficult to handle that proper treatments are not attempted. Abscessed teeth are neglected so long as the child is free from pain; or if the child is suffering, nothing further than is necessary for temporary relief is undertaken. Every dentist should exert himself to carry through treatments of the deciduous teeth to a really successful result.

Another point of still greater importance perhaps is the awakening of a feeling among the laity and especially among the parents that children should visit the dentist often, in order that simple operations may be done in ample time to prevent the development of more serious conditions. By this plan the child will have less fear and will come to have confidence that the service of the dentist will be of great benefit. Children are quick

to appreciate a successful operation. The dentist should continually impress upon the parents the necessity of care of their children's teeth; correcting misapprehension and giving information as to the means of cleaning for the prevention of decay, as far as that is possible. The dentist should enter into close relation with mothers and nurses upon these subjects, in order that they may work together understandingly for the benefit of the children. Until this is done many children will be brought to the dentist for the first time with aching teeth, with all of their sensitiveness developed, and it will be impossible to handle them properly.

Filling of deciduous teeth. In the deciduous molar teeth decays in the occlusal surface often occur very early. At the first opportunity after the eruption of these teeth the occlusal surfaces should be carefully examined, and if the pits and fissures are deep, though not decayed, these should be filled with cement at once, without cutting cavities. This is best done perhaps with oxyphosphate of copper cement, filling them as full as the occlusion will allow. Such fillings will require renewal from time to time.

If the child presents after decay has occurred, the decay should be very thoroughly removed and fillings made. The copper cement is being used extensively for this, and is one of the most satisfactory materials available. Care must be taken to remove every particle of carious dentin, otherwise the tooth is liable to be discolored. Fillings of base-plate gutta-percha, especially in occlusal cavities, will usually not last quite as well as those of copper cement. Amalgam should not be used unless conditions are such that the cavity may be unquestionably dry. If the cavity walls are moist in the slightest degree, a recurrence of decay is practically certain and this will often progress to the involvement of the pulp before it receives attention. Copper cement or gutta-percha do not permit such a leakage, but are gradually cupped out by wear. They should be replaced as often as may be necessary. Care should be taken to maintain contacts between the deciduous molars, and between the second deciduous molar and the first permanent molar, to prevent the impaction of food.

The deciduous incisor teeth begin to separate as the arch expands with the growth of the permanent teeth. Usually the movement begins during the third year, after which fillings may be made in proximal surfaces of these teeth without the neces-

sity of restoring contacts. In my work on Operative Dentistry I have called attention to the plan of cutting away slight proximal decays of these teeth which have occurred before the teeth have begun to separate. In this way further decay may be prevented and the necessity of placing fillings avoided.

These few suggestions relative to the management of caries in the deciduous teeth are made for the especial purpose of emphasizing the importance of the care which is necessary to protect their pulps.

ACUTE ALVEOLAR ABSCESS

ILLUSTRATIONS: FIGURES 402-429.

AN abscess is a collection of pus in a cavity formed within some tissue or organ of the body as a result of suppuration. An alveolar abscess is so named because it occurs within the bone which forms the alveolar process.

I have previously described the lateral alveolar abscess, which also involves the peridental membrane and the bone of the alveolar process. Such an abscess occurs as a result of an inflammation which originates at the gingival line of the tooth and the pulp may be alive or dead; there is no relation between the condition of the pulp and the formation of a lateral abscess. The occurrence of true alveolar abscess is always subsequent to the death of the pulp.

It might seem that a modifying adjective should be used to more clearly designate a true alveolar abscess.* The term alveolar abscess has been so generally used to designate this condition that I am inclined to favor its continuance, and to make the differentiation of the other abscesses of the investing tissues of the teeth by using the qualifying terms in designating the positions in which they occur. These are the gingival abscess, septal abscess and lateral alveolar abscess. In this writing we will define alveolar abscess as a collection of pus in the periapical tissues due to infection from the root canal of a tooth.

Etiology.

As a sequel to the destruction of the pulp by hyperemia or inflammation, no matter how these are caused, the area of peridental membrane around the apex of the root may become involved in inflammation. This occurs only after the pulp in the tooth is dead from one of the causes mentioned. Following the death of the pulp, there are three principal exciting causes of periapical inflammation: (1) The escape through the apical foramen of saprophitic organisms, or the poisonous products of

* Dr. Thomas L. Gilmer, in a paper on Alveolar Abscess, in the Dental Review, Vol. 28, 1914, p. 427, has used the term *typical alveolar abscess* to describe those abscesses following the death of the pulp, and *atypical alveolar abscess* as applying to those which occur from infections of the peridental membrane not due to the death of the pulp.

putrefactive decomposition; (2) the escape of pyogenic organisms or the products of suppuration of the pulp; (3) the passage of certain medicaments placed in the root canal in treatment. The affection is not materially different because of the different conditions under which the pulp may have died, but is perhaps more rapid in its development in the cases in which the pulp has died from suppuration, for in such cases the apical tissues are infected directly by the material passing from the pulp into the apical space.

Pathological changes.

Apical pericementitis and pus formation. The first involvement of the periapical tissues is an apical pericementitis; when such an inflammation progresses to the stage of pus formation, it becomes alveolar abscess. Most cases progress rapidly to pus formation, although occasional cases present in which the periapical inflammation continues for an indefinite period without suppuration.

Absorption of bone. An area of bone forming the walls of the alveolus of the tooth is involved by the inflammatory movement, and absorption of the bone quickly occurs. The fibers of the peridental membrane of the region elongate as this absorption takes place, extending out to and perhaps maintaining some connection with the bone which is being absorbed, producing a fan-like projection of comparatively large fleshy fibers extending out from the apical end of the root to the absorbed area. Between and among these fibers is the accumulation of pus, rather than in distinct abscessed cavities. As the suppuration proceeds, widening its area in the bone, more definite abscessed cavity forms may appear.

The pus soon penetrates the dense plate of bone forming the socket and enters the cancellous bone. (See Figure 471.) The latter is easily penetrated on account of the many open spaces. After a few hours more time, the pus will find a way to the surface of the bone, usually nearest the point of the apex of the root, through some Haversian canal which offers an easier exit than another similar canal in the neighborhood, and this is enlarged by absorption, affording an exit of the pus to the surface of the bone, under the periosteum. (See Figures 402 to 416, also 430 to 432.)

The extent of the area of the absorption of bone varies, and may occur rapidly in some cases, and slowly in others. I have seen cases in which all of the changes thus far described occurred

FIGS. 402 TO 407. A series illustrating acute and chronic alveolar abscess occurring in the upper incisor region.

FIG. 402. Blind abscess at the root of an upper central incisor: a, Abscess-cavity in bone. b, Drill-hole exposing the pulp chamber for treatment.

FIG. 403. Acute alveolar abscess of upper central incisor pointing on the gum: a, Abscess cavity in the bone. b, Floor of the nostril. c, Lip. d, Tooth. In this case the pus has penetrated the periosteum without lifting it from the bone.

FIG. 404. Chronic alveolar abscess at the root of an upper incisor with sinus discharging on the gum: a, Abscess cavity in the bone. b, Mouth of sinus. c, Lip. d, Tooth.

FIG. 405. Acute alveolar abscess in which the pus has lifted the periosteum from the bone: a, Abscess cavity in the bone. b, Floor of the nostril. c, Lip. d, Tooth. e, Pus cavity beneath the periosteum.

FIG. 406. Acute alveolar abscess, the pus from which has raised the periosteum from the hard palate: a, Very large abscess cavity in the bone. b, Pus cavity between the periosteum and bone. c, Lip. d, Tooth. e, Floor of nostril.

FIG. 407. Alveolar abscess at the root of an upper incisor discharging into the nose: a, Large abscess cavity in the bone. b, Mouth of sinus on the floor of the nostril. c, Lip. d, Tooth.

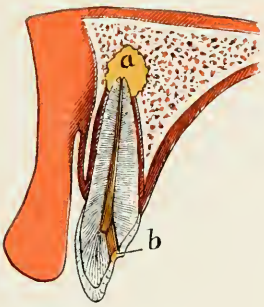

Fig. 402.

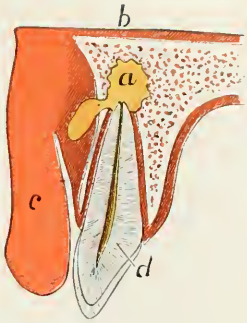

Fig. 403.

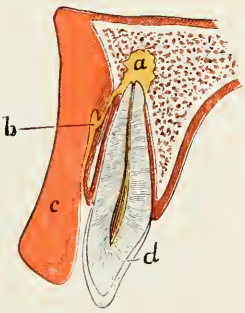

Fig. 404.

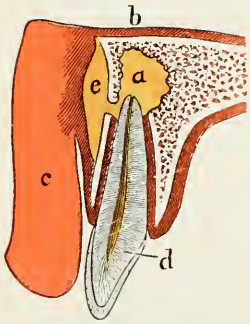

Fig. 405.

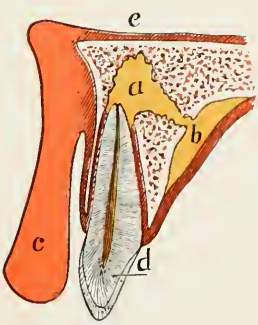

Fig. 406

Fig. 407.

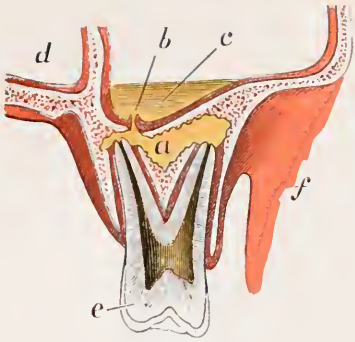

Fig. 408.

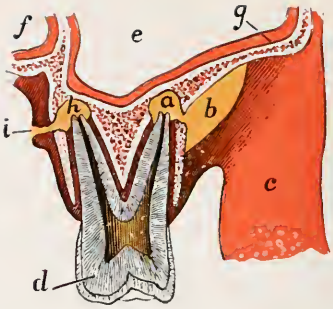

Fig. 409.

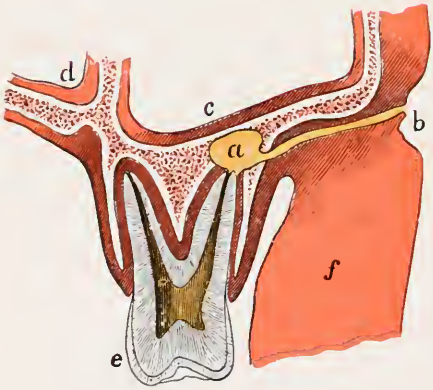

Fig. 410.

Fig. 411.

FIGS. 408 TO 411. A series illustrating acute and chronic alveolar abscess occurring in the upper molar region.

FIG. 408. Alveolar abscess at the root of an upper molar discharging into the maxillary sinus: a, Abscess cavity in the bone. b, Mouth of sinus in the floor of the antrum. c, Pus in the antral cavity. d, nasal cavity. e, Tooth. f, Tissues of cheek.

FIG. 409. Acute alveolar abscess from the buccal roots and chronic abscess from the lingual root of an upper molar: a, Cavity of acute abscess in the bone. b, Pus cavity between the bone and periosteum, extending out under the prominence of the malar process. c, Tissues of cheek. d, Tooth. e, Maxillary sinus. f, Nasal cavity. g, Malar process. h, Cavity of chronic abscess discharging at i.

FIG. 410. Alveolar abscess from the buccal roots of an upper molar discharging on the face: a, Abscess cavity in the bone. b, Sinus opening on the face. c, Maxillary sinus. d, Nasal cavity. e, Tooth. f, Tissues of cheek.

FIG. 411. Scar caused by alveolar abscess discharging on the face.

over night, and the face had begun to swell in the morning, while in others a week elapsed before the bone was penetrated.

If pus penetrates periosteum. When the pus has forced an exit through the bone and periosteum and enters the softer tissues, it will usually form a rounded tumor, occupying the center of an area of infiltration formed to wall it off. Generally within from one to three or four days, if the tissues are not incised, the pus will find an exit through the soft tissues and be discharged. (See Figures 403 and 412.)

If pus lifts periosteum from bone. In a minority of the cases, the pus, on arriving at the surface of the bone, meets with conditions in which the periosteum is raised from the bone, and, instead of penetrating the periosteum, the pus spreads out in a broad, flat tumor between the bone and the periosteum, as distinguished from the rounded tumor previously described. (See Figures 405, 406, 409 and 413.)

This flat form of tumor over the affected area in alveolar abscess is very much the more dangerous form, for it is liable to involve the bone in necrosis. Necrosis occurring from alveolar abscess has practically always been in the cases in which the periosteum was lifted from the bone and this broad, flat swelling occurred. Occasionally, the periosteum will build a plate of new bone in its new position. This is called an *involucrum*. It may enclose the necrosed portion. Figure 463 is of a boy who suffered from a necrosis of the lower jaw on the left side and a large involucrum was so formed. His cheek, which is apparently much swollen, is of the contour shown in the illustration, because of the building of the new subperiosteal bone in that position.

Variations in burrowing of pus. These cases, when left to themselves, may present some very peculiar features. Pus may become entangled among the fascia and burrow to considerable distances. In the upper jaw it may raise the periosteum along the prominences of the malar process, or the malar bone itself, and discharge upon the face. (See Figure 411.) Or it may take other directions and discharge upon the face. Or again it may discharge on the gum similarly to the more ordinary form of alveolar abscess, or it may tear up the periosteum to the gingiva, and discharge between the gingiva and the tooth. In fact, in this class of alveolar abscess, location of the discharge is very variable, while in that class which gives the ball-like form of tumor, the location of the discharge is almost universally upon

the gum in the immediate neighborhood of the tooth. (See Figures 402 to 416.)

DISTINCTIONS BETWEEN ALVEOLAR ABSCESS AND ABSCESSES OCCURRING ELSEWHERE.

Alveolar abscess has characteristics which distinguish it prominently from all other abscesses occurring in the body. It is an abscess which has to do especially with the bones of the jaws and is formed at the ends of the roots of the teeth in the depths of the bony tissue. It is the bone which seems to suffer most, as is shown by the pus cavity forming within the bone. The peridental membrane, which is the real seat of affliction, is usually only slightly involved in the formation of the abscess cavity.

INFECTION FROM PULP CHAMBER OF A TOOTH. The first and most important distinguishing feature is the fact that the infection which causes this abscess is derived from the pulp chamber of a tooth after the pulp has died. This infection will continue so long as the infected material remains in the root canal. It is for this reason that the abscess, after the acute stage has passed, takes on a chronic character. Practically all acute abscesses become chronic if not treated, because of this continual reinfection from the contents of the pulp chamber. This is a character found in alveolar abscess which occurs in no other part of the body and requires special technical treatment. The tooth affected must have proper preparation of the root canal and the apical foramen, and be completely and perfectly closed by a filling which is placed in it for this purpose, before the abscess can get well.

CEMENTUM, IF DENUDED, MAINTAINS CHRONICITY. In some of the cases, necrosis of bone occurs in connection with the formation of the abscess, the same as necrosis of bone occurs in other regions in the body, resulting from abscesses in connection with or in the bone. There is no particular difference in this part of the injury from that which occurs in other abscesses involving bone. The necrosed bone is separated from the living bone by absorption, it loosens, and finally the sequestrum may be removed. As in abscesses in the bone elsewhere, it is required that every particle of necrosed bone be removed before the abscess can get well, but in alveolar abscess there may be still another element which does not appear in necrosis of bone elsewhere. Sometimes fibers of the peridental membrane, covering the root end or some portion upon the side of the root, may be destroyed during the

FIGS. 412 TO 416. A series illustrating acute and chronic alveolar abscess occurring in the lower incisor region.

FIG. 412. Acute alveolar abscess from lower incisor pointing on the gum: a, Abscess cavity in the bone. b, Sinus opening. c, Lip. d, Tooth. In this case the pus has penetrated the periosteum without lifting it from the bone.

FIG. 413. Acute alveolar abscess from lower incisor with pus cavity between the bone and the periosteum: a, Pus cavity in the bone. b, Pus between the periosteum and bone. c, Lip. d, Tooth.

FIG. 414. Chronic alveolar abscess at the root of a lower incisor: a, Abscess cavity in the bone. b, Sinus discharging on the gum. c, Lip. d, Tooth.

FIG. 415. Chronic alveolar abscess at the root of a lower incisor with sinus discharging on the face under the chin: a, Abscess cavity in the bone. b, b, b, Sinus following the periosteum down to the lower margin of the body of the bone and discharging on the skin. c, Lip. d, Tooth.

FIG. 416. Chronic alveolar abscess at the root of a lower incisor with abscess cavity passing through the body of the bone and discharging on the skin beneath the chin: a, Very large abscess cavity. b, Mouth of the sinus.

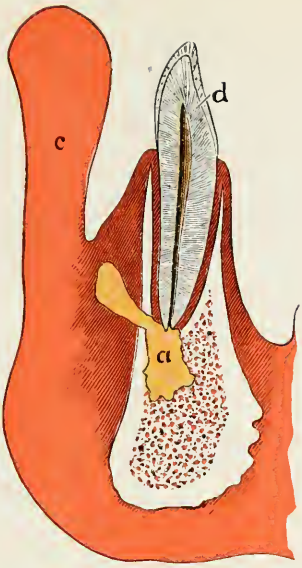

Fig. 412.

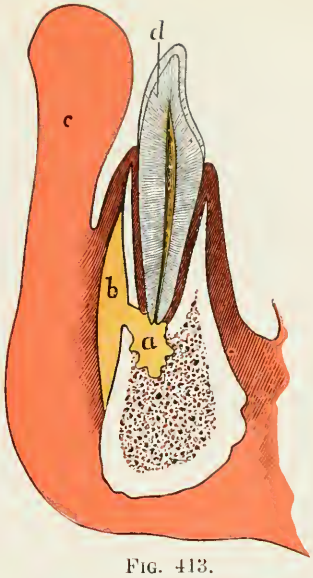

Fig. 413.

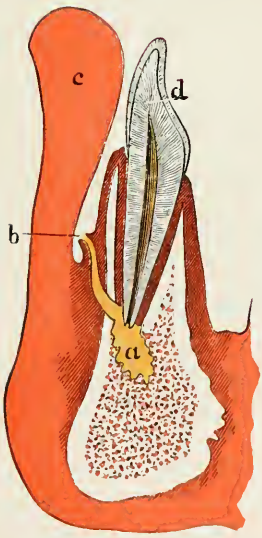

Fig. 414.

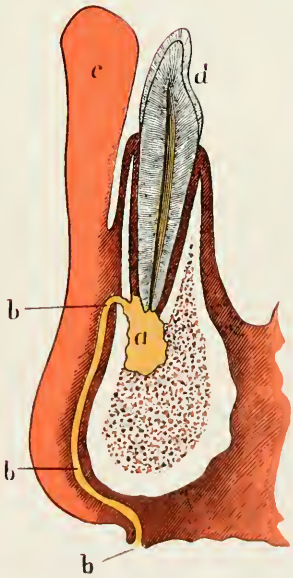

Fig. 415.

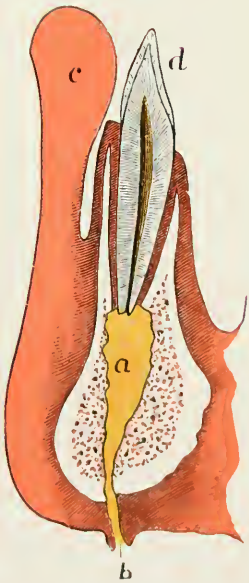

Fig. 416.

Fig. 417. July 27.

Fig. 418. Aug. 6.

Fig. 419. Sept. 29.

Fig. 420. Nov. 10.

Fig. 421. Dec. 22.

Fig. 422. Feb. 1.

Figs. 417 to 422. A series of radiographs of a case of acute alveolar abscess from an upper cuspid. A distal gold filling was placed in this tooth on December 23, 1913, the pulp being vital at the time. The tooth remained comfortable until July 27, 1914, when the patient presented with an acute abscess, which was lanced at once, and a radiograph taken; Figure 417. The root was filled August 6, when the second radiograph, Figure 418, was taken. The other radiographs were made on the dates mentioned, showing the gradual building in of the bone. There is some question whether or not this case will fully heal, as it is probable that the pulp died soon after the filling was placed and a blind abscess may have developed without symptoms, destroying the tissues about the apex previous to the occurrence of the acute abscess. Case from practice of Dr. Arthur D. Black.

acute inflammation and the life of that portion of the cementum is lost and the part denuded becomes pus soaked. This pus-soaked cementum then serves to maintain a chronic abscess, just the same as would a spicula of necrosed bone, with the difference that the pus-soaked area of cementum can not be exfoliated, as would be the case with the piece of dead bone.

These two items of difference between alveolar abscess and other abscesses of the body render it a special form of abscess, presenting interferences to the healing process which require special technical treatment.

Symptoms.

The general rule is that periapical inflammations of the peridental membrane are short and decisive, presenting characters of rapidly growing intensity, running their course to suppuration within a few hours. The progress from the condition exhibiting no symptoms whatever to a definite acute alveolar abscess may be so rapid that the intermediate state of apical pericementitis will not be recognized; or the inflammation may be of such a low grade from the first that a chronic alveolar abscess will be formed and exist for a considerable time — possibly for years — without symptoms which will have in any way attracted the attention of the patient.

Constitutional symptoms. The constitutional symptoms usually consist of a sudden rise of temperature, often ranging from 102° to 105°, with a correspondingly rapid pulse. In a few cases I have seen a temperature of 106°. There may be a chill preceding the fever. The patient may complain of headache, malaise and other symptoms commonly accompanying an acute infection. A blood count will show a leucocytosis.

Local symptoms. The local symptoms of acute alveolar abscess are: (1) More or less soreness of the tooth; (2) a slight protrusion of the tooth from the alveolus; (3) tenderness of the tooth to use in mastication, or to the touch of the teeth of the opposite jaw, or to percussion; (4) sometimes looseness of the tooth; (5) pain, which may be very persistent and of a dull character, or which may increase very rapidly in severity; (6) there may be a general hyperemia affecting more or less of the gum tissue, especially on the labial or buccal side, where the tissues are thinnest; (7) swelling of the neighboring tissues; (8) an accumulation of pus indicated by fluctuation.

Tenderness of tooth. The most prominent early symptom is likely to be the soreness of the tooth. It will be tender to the

touch, it may be slightly protruded by the inflammation in the periapical space. The inflammation may cause a relaxation of the fibers of the peridental membrane so that the tooth is loose in its socket. There is generally no redness or swelling of the gum tissue at this time.

Pain and swelling. When pus formation occurs rapidly, there is an increasing soreness which leads to a throbbing character of the pain. The peridental membrane of the region, while a little thicker than elsewhere, is only a small amount of tissue, which lies between the apex of the root and the walls of the alveolus. In this confined space the sharp inflammatory movement grows worse, and the pain becomes intense, being increased by every pulse beat. If the case is running a rapid course, fever will develop at this stage and increase in severity as the inflammatory movement and pus formation increases. The pain may become almost intolerable.

When the pus has forced an exit through the periosteum to the soft tissues covering it, the pressure within the bone is relieved, and almost immediately the intensity of the pain diminishes and more or less swelling occurs, with marked extensions of the hyperemic movement all about it, reddening the surface generally; so that patients often report that the pain ceased when the face swelled, and yet that is not quite true. It is a fact, however, that the pain is usually greatly moderated just at this juncture. In a few cases the swelling at this time will become enormous. If the case be in the upper jaw, it is not very unusual for the eye of that side to be closed, and the patient unable to open it because of the swelling of the lids and of the soft areolar tissue about it. In some of the severer cases, the swelling may involve almost all the tissues of the side of the face. If the abscess occurs in the lower jaw, the upper face will not swell so much, but the swelling will involve the floor of the mouth and the tissues of the cheek and of the angle of the neck, rather more than the tissues of the face, and present a greater tendency for the pus to be discharged upon the face or neck. It is usually during this swelling of the soft tissues that the temperature is highest. Cases, however, vary from this ugly picture to the milder forms which present little or no swelling, and little or no pain. Between these two, any variety of rapidity of progress of inflammatory movement, and of fever or lack of fever, may occur, possibly continuing for several weeks.

Ball-like tumor. When the pus escapes from the bone and passes through the periosteum into the connective tissues, an

examination with the finger will discover a ball-like tumor, within which an area of fluctuation may be made out. This tumor is inclined, in the great majority of the cases, to point on the gum somewhere in the neighborhood of the nearest approach to the root of the tooth involved, and will, if left to itself, make an opening upon the surface of the gum tissue and discharge its pus into the mouth. (See Figures 403 and 412.)

Flat tumor. In cases in which the periosteum is not penetrated, but is stripped from the bone and the pus accumulates between it and the bone, the tumor is broad and flat. It is more difficult to detect the presence of pus by palpation, on account of the tenseness of the periosteum. Failure of proper diagnosis at this stage inclines to the postponement of surgical interference, and necrosis of the involved bone frequently results. (See Figures 405, 406, 409 and 413.)

Painful symptoms disappear with discharge of pus. As soon as the pus has been discharged from either of these forms of abscesses, the painful symptoms disappear almost at once. A lingering soreness continues for some days, the swelling disappears and the patient becomes comparatively free from subjective symptoms, but the abscess does not get well. The pus becomes less and less until only a small amount is discharged. The tooth can be used in mastication the same as before and resumes its normal work, but there is still a continuous discharge of pus from the opening on the gum, or from the tissues wherever an opening has become established, whether it be on the face or elsewhere. This is chronic alveolar abscess, which will be described later.

The symptoms of amelioration which mark the change from the acute to the chronic variety of alveolar abscess do not admit of specific designation as to when the acute variety is ended, and the chronic form has become established. We generally consider the chronic form established, however, when the tooth is well enough to return to its ordinary work of mastication.

Differential diagnosis between acute alveolar abscess and certain other conditions.

While there is usually little difficulty in making a positive diagnosis in cases of acute alveolar abscess, there are several conditions which may present symptoms which will require a differential diagnosis. The most important of these are certain tumors, more particularly sarcoma and gumma, also aneurism, cysts, and glandular enlargements. It is not within the scope of

this book to go into detail in the differential diagnosis of these conditions, but to call attention to them and mention the most important differential symptoms. In all cases in which there is any question as to the diagnosis, careful inquiry should be made into the history, as a basis for a proper and thorough examination.

Sarcoma. Sarcoma will be differentiated by the fact that the swelling is of slower development than in cases of abscess, usually without painful symptoms, and the enlargement is felt to be a solid mass of tissue without fluid contents. In some cases of alveolar abscess, however, particularly if the pus has lifted the periosteum without having penetrated it, it may be impossible to detect fluctuation. A radiograph of a giant cell sarcoma of the lower jaw is shown in Figure 423.

Gumma. Syphilitic gumma may occur about the mouth in positions in which alveolar abscesses occur, more frequently in the upper jaw. These are also of slower development, usually without pain, and the mass is recognized as a solid tissue growth. In such cases there are other symptoms of syphilis, which will be elicited by further inquiry into the history and a Wasserman test. While the Wasserman reaction is generally to be relied upon, it should be remembered that occasionally a case known to be syphilitic gives a negative Wasserman.

Aneurism. An aneurism of the posterior palatine artery might be mistaken for an alveolar abscess which had penetrated the bone of the palate. This should be differentiated by the absence of fever and pain, by the fact the tumor may be reduced by pressure, and by the pulsations. It might be difficult to control the hemorrhage from an aneurism in this position if the tissues should be incised. However, aneurism of the posterior palatine artery is rare.

Cysts. Cysts within the maxillary bones occur quite frequently. These may present as fluctuating tumors. They are of gradual growth, without inflammatory symptoms, and usually without pain. A radiograph will usually show a smooth regular outline to the cavity, indicating the pressure destruction of the bone, as compared with a more irregular destruction in abscess formation. The fluid of the cyst is clear, of yellowish color, odorless, and viscid; it may be drawn out into strings of considerable length. The subject of cyst formation in connection with alveolar abscess is treated elsewhere. Figures 424 and 425 are radiographs of cysts, and Figure 426 is a photograph of a

skull showing a cyst cavity. The smooth outline of these cyst cavities will be noted.

Examine fluid contents. In all tumors containing fluid in which there is uncertainty as to the diagnosis, some of the contents should be withdrawn with an aspirating needle, or the tumor may be punctured with a grooved needle, and by turning this back and forth, some of the contents will escape along the groove.

Glands. The submaxillary or sublingual glands may be enlarged on account of obstructions of their ducts, or as a result of infections involving the glands. The cervical lymphatic glands may be enlarged as a result of infections anywhere in the region which they drain. When the cervical lymphatics are found enlarged, careful search should be instituted and continued until the cause is learned.

Eruption of third molars. Inflammations often occur in connection with the eruption of the third molars, and especially as a result of abnormal positions of these teeth. It is sometimes difficult to differentiate these from alveolar abscess, because it may be impossible to open the mouth sufficiently for a thorough examination. In many cases a radiograph will be of material assistance. If the mouth can not be opened to permit the use of a small film, this region of the angle of the jaw can be well shown on a plate by having the direction of the rays such as to miss the jaw of the opposite side.

Lateral alveolar abscess, septal abscess and gingival abscess, together with the points of differentiation from true alveolar abscess, have already been mentioned.

In all cases, a critical examination of the teeth of the region is important. If they respond to tests for pulp vitality, alveolar abscess is excluded. It is of course recognized that teeth which do not respond may have had the pulps removed and may be excluded by radiographs; also that teeth which have dead pulps may not have caused an infection of the periapical tissue. Several of the conditions mentioned are not frequently observed by dentists and, whenever there is doubt, a physician or surgeon should be called in consultation. No case should be dismissed until every means to a proper diagnosis has been employed.

As illustrating the need of careful differential diagnosis, I cite the following cases reported by Dr. Thomas L. Gilmer in

a paper* on alveolar abscess read before the Chicago Dental Society.

"First case: Patient, woman, forty years of age. There was extensive swelling under the mandible near the angle, which extended forward and up the cheek. There was a slight trismus, which prevented complete opening of the jaws; temperature 102°; there was much pain and general discomfort.

"On examining the teeth I found all of the pulps alive on this side of the mouth. The third molar had been removed fifteen years before, therefore infection from an impacted tooth was excluded. There was no indication of a lesion in any part of the mouth or on the face. The tonsils were normal and no previous history of tonsilitis, or cervical or submaxillary lymphadenitis could be elicited. Owing to the swelling, it was difficult to palpate the submaxillary salivary gland. No stone was found in Wharton's duct, but the tube was enlarged and hard. Pressure upon the submaxillary gland and stripping of Wharton's duct caused pus to flow from the duct. Here was the secret of the clinical manifestations. It was an infection of the submaxillary gland, which had caused the condition, and gave some of the symptoms of acute alveolar abscess.

"Second case: Patient, boy, 14 years of age. He was assigned to my service at St. Luke's Hospital, after diagnosis of alveolar abscess had been made. On examination of the patient, I found a large, red, shiny swelling extending from the neck up to near the malar bone, from back of the angle of the mandible forward to the region of the first lower bicuspid. There was slight pain; temperature 100°; blood showed 14,000 leucocyte count.

"Owing to the great swelling I was unable to palpate any of the glands under the jaw, or in the cervical region. Wharton's duct appeared normal. The history elicited the information that the swelling came on rather slowly, ten days since it had first been noticed. There was occasional trismus, but the jaw could be sufficiently opened to make a careful examination of the mouth and teeth. All of the permanent teeth were in place except the third molars. Those erupted were sound and none of them were sore to the touch. The teeth were cleaner than is usually found in the mouth of such a child. The pulps in the teeth all responded to the faradic current test. The teeth were excluded.

* Etiology, Diagnosis and Treatment of Acute and Chronic Alveolar Abscess, Dental Review, Vol. 28, 1914, p. 427.

FIG. 423.

FIG. 424.

FIG. 425.

FIG. 423. Radiograph of a case of a giant cell sarcoma in the region of the lower cuspid and first bicuspid.

FIG. 424. Radiograph of a case of a cyst of the lower jaw, boy twelve years old. The smooth outline of the area of bone destroyed is shown.

FIG. 425. Radiograph of a case of a cyst in the upper bicuspid region. The apices of the bicuspid roots have been forced apart by the pressure exerted by the cyst.

Fig. 426.

Fig. 426. A lower maxilla showing a smoothly rounded cavity, evidently made by a cyst. Specimen from Northwestern University Dental Museum.

The tonsils were enlarged, red and somewhat ragged. The child said that he had often had sore throat; that twice before he had a slight swelling under the mandible, and that preceding this attack, and also preceding the previous attack, he had felt "kernels" (enlarged lymph nodes) under the jaw.

"Diagnosis: Suppurative lymph-adenitis, secondary to tonsilar infection. On palpation I thought I was able to make out very deeply in the tissues a slight fluctuation. I incised the skin and made blunt dissection, liberating a considerable quantity of creamy pus."

TREATMENT OF ACUTE ALVEOLAR ABSCESS.

ILLUSTRATIONS: FIGURES 427-429.

It must be held distinctly in mind that the forerunning condition which produces alveolar abscess is the death of the pulp of the tooth. The infection which occurs in the destruction of the pulp by suppuration and saprophytic decomposition passes through the apical foramen, infecting the tissues in the apical space. In some cases this inflammation may be slight, but in most cases it passes quickly into pus formation.

Usually the organisms enter the pulp tissue through a cavity of decay in the tooth which has exposed the pulp, although they doubtless reach the pulp in a limited number of cases through the blood stream. There is much evidence now that micro-organisms may remain in the tissues for months and even years and then be brought by the blood stream to a point of injury and there set up an infection. Thus there may be pus formation in the apical space after the death of the pulp in a tooth in which there is no cavity.

During apical pericementitis.

The first object in treatment would seem to be the shutting off of the ingress of micro-organisms through the apical foramen, but this would be impracticable in the larger proportion of cases until after the acute symptoms have subsided. However, should the case present during the stage of apical pericementitis, or when there is hope of aborting the formation of an abscess, the root canal should be cleaned at once.

In such cases, some form of counter irritation should be employed. I have usually preferred to saturate a bit of gauze or cotton, small enough to be covered by the finger, in chloroform and, after absorbing away the excess, place it upon a piece of rubber dam, a little larger than can be covered by the finger, and apply this to the gum over the root of the tooth affected. It should then be held with the finger until a sharp burning of the tissue occurs. Then it may be removed for a time and reapplied. This may be done several times at one sitting, possibly producing a blister. It has seemed to me that this treatment gives better results if it is stopped short of forming a blister.

Oftentimes this will produce at least temporary relief. Various other forms of counter irritation have been used, such as cantharides plasters, and other like irritants. A saline cathartic should be regularly ordered, also a hot foot bath before the patient retires. All of these are palliative rather than curative.

The tooth may be given rest by building up the occlusal surfaces of other teeth with cement. I prefer the oxyphosphate of copper cement for this purpose, because it adheres better, applying a thin layer on the occlusal surfaces of the bicuspids and molars of both sides of one arch, except of course in the position of the tender tooth.

SECURE GOOD DRAINAGE.

Most cases of acute alveolar abscess present with a sore tooth, the soreness having developed very recently, and it is found that the pulp of this tooth is dead — the pulp chamber may be open or not — and the case is becoming worse from hour to hour. The first treatment should be to obtain drainage for the pus and relieve the pain. The conditions in the particular case will indicate the plan of treatment to be followed. There are two routes by which the abscess cavity may be reached: (1) through the root canal, and (2) through the gum and alveolar process, or in some cases externally on the face or neck.

THROUGH PULP CHAMBER. If the tooth is not too sore, and if the case has not progressed to the stage when a tumor containing pus may be palpated, an attempt should be made to open the pulp chamber sufficiently and clean it and the root canals. Sometimes fairly thorough mechanical cleaning of the root canals, especially if the canals are large enough to permit a broach to be passed through the apical foramen, will give relief, by thus giving drainage through the canals. When this is done the rubber dam should be in place, and, after the canals have been cleansed, a dressing should be securely sealed, as has been described in the technic of pulp treatment.

THROUGH INVESTING TISSUES. The other plan, which should be more generally followed, is to make an incision through the gum for the discharge of the pus, leaving the treatment of the pulp chamber and root canals until immediately after the acute symptoms have subsided. In those cases in which a fluctuating tumor may be palpated, whether the pus be under the periosteum or outside of it, an incision is positively indicated as a first procedure, regardless of the condition of soreness of the tooth.

In those cases which present previous to the formation of a

palpable tumor, but with the tooth too sore to justify an attempt at treatment through the pulp chamber, and also in cases in which treatment through the pulp chamber has been tried without securing reasonably prompt relief, one of the very best expedients is to make a considerable cut through the gum tissue to the bone as near the apical end of the root as is practicable. This will differ with different teeth. In making this cut, the edge of the knife should be pushed to one side and then to the other, in order to tear up the periosteum from the bone over a space.

Advantages of early incision. Several advantages will often be gained by such an incision. The hemorrhage will relieve the congestion and, if a slight amount of pus should have penetrated the bone and reached the periosteum, relief will be secured. If the pus has not yet reached the outer plate of bone, the opening will very likely be found by it within a few hours, and the duration of the pain will thus be cut short. The possibility that the periosteum will be lifted from the bone will be avoided and necrosis will often be prevented. There seems therefore to be every reason for an early incision to and through the periosteum. It would be much better if earlier incision were made in practically all cases. Even though such an incision should be occasionally made when not absolutely necessary, no harm would be done. My observation of hundreds of cases in which serious complications have occurred as the result of failures to make incisions early, or not at all, leads me to strongly emphasize the desirability of more prompt surgical interference in these cases.

Incision should be ample for good drainage. In opening an abscess, an incision should be made that will give very free exit to the pus. The incision should usually be from three-fourths of an inch to a full inch in length. If a very small cut is made, good drainage is not secured, and a second incision will be necessary. There is practically no difference in the pain caused in making a slight or a liberal opening.

Operators differ as to the best direction for the incision. Some make it high on the process, parallel with the length of the jaw. It has been my habit to enter the lance high up, stretching the mucous membrane away for that purpose, and make the incision parallel with the long axis of the tooth, as far as midway of the root or a little farther. Then, by moving the end of the blade to one side and then to the other in this cut, the periosteum may be cut or torn away from the bone for a little distance.

If the pus flows freely about the knife, this lateral motion of the instrument may be omitted.

If a broad, flat tumor under periosteum. In case we find a broad, flat fluctuating tumor which is formed by the pus tearing up the periosteum and remaining between the periosteum and bone, a very broad opening should be made. It is from this class of cases that necrosis of bone as a sequela of alveolar abscess practically always occurs and the drainage afforded should be such that the pressure will be unquestionably relieved and the periosteum will drop back to its proper place. (See Figures 405, 409 and 413.)

If pus has not reached the periosteum. In those cases in which the pus has not reached the periosteum, following the incision through the soft tissues, an opening may be made through the bone to the region of the apex of the root. This may be done with a sharp bibeveled drill in the engine. This procedure may be employed in only a limited number of cases when the access is good. The severe pain will be relieved in a very short time. The operation itself, however, is often very painful. The greatest care should be taken not to cut away the peridental membrane from any part of the root, as this might produce conditions which would render the abscess incurable.

There should be no hesitancy in enlarging openings in the bone, if sufficient care is taken to avoid injury to the peridental membrane. My experience has been that there is no tissue which heals much more kindly than does bone, and cutting the opening larger seems to make but little difference in the healing process.

Anesthesia for incision. The incision and the cutting of the bone are both very painful, although of but a moment's duration. The administration of nitrous oxid is the best means of securing anesthesia. The use of novocain locally in these cases is generally not satisfactory.

Opening made with phenol. I have followed another plan in an occasional case which was slow in developing and in which there was little swelling with considerable pain. An instrument fashioned like a plugger, but with longer and sharper serrations, is dipped into phenol and, after being withdrawn, is held until there is a single small drop of phenol on the end of the instrument. The area is protected by cotton rolls, and the phenol on the end of the instrument is brought to the spot nearest the apex of the root, and applied. At once it whitens a small area about it. When this has occurred, the tissue that is whitened is

removed by a scraping motion of the plugger point. Then another drop of phenol is applied in the same way, and the scratching of the tissue with the instrument is repeated. The drawing of blood by the scratching of the instrument should be a signal to clean out what has been done, and apply more phenol before proceeding. An opening is thus gradually made through the soft tissues to the bone. The walls of this passage are so benumbed by the phenol that they may be stretched wider open, and some little cutting may be done without pain, to enlarge the opening. Then with a blunt instrument the periosteum may be raised about the opening, and the bone may be penetrated as detailed above. A very nervous patient, who dreads an incision, may appreciate this method, although it requires quite a little time.

Irrigation. Following the incision, it has been my habit to very thoroughly irrigate the wound with salt solution, using for this purpose either the large rubber bulb syringe or a fountain syringe. This has always been done with a syringe point having a sufficiently large opening to permit of a free flow of the solution without much pressure, in order to secure the most thorough cleansing and at the same time avoid the danger of forcing the infection deeper into the tissues. It is also necessary that the wound be well opened, to give opportunity for the free return flow of the fluid, otherwise an extension of the infection might result.

During recent years the tendency among surgeons has been to make very free incisions, and omit the irrigation, relying upon packs moistened frequently with boric acid or salt solution to keep the wound patulous. As a general proposition this plan seems to be giving better results, although it applies more especially to hospital rather than ambulatory cases. There is also a greater tendency for incisions within the mouth to close, as compared with those made through the skin.

Packing. In no case should the precaution to keep a free opening through the soft tissue be neglected, for in a great many cases such openings heal very promptly. I have seen broad cuts, through which pus escaped freely, united within six hours so that it was necessary to again incise the tissues. It should be remembered that the gum tissue heals very quickly. This is one of its characteristics.

The opening in the soft tissues may be maintained by packing in a small strip of gauze. This should not be packed tightly enough to interfere with the drainage. The gauze may be first

saturated in 95 per cent phenol, and then pressed between the folds of a sterile towel until the gauze or cotton is practically dry. This should be carried to the depth of the cut and should remain twenty-four or forty-eight hours. This slight amount of phenol on the gauze serves to keep the opening from closing as rapidly as it otherwise would. It is not used with the intention that the phenol will, by its antiseptic property, promote the healing of the abscess, but to retard the normal activity of the freshly cut surfaces toward reuniting. The gauze should not be carried into the bone cavity.

Open pulp chamber after acute symptoms have subsided.

During the time of severe soreness of the tooth, no effort should be made to open the pulp chamber. The tooth is tender; every touch upon it hurts, and there is no advantage from such interference. The incision through the gum will relieve the patient of pain earlier than any other treatment, and the opening of the tooth and cleaning of the root canals should be deferred until the acute symptoms have subsided. Then these operations can be performed without pain. Figures 417 to 422 are reproductions of radiographs of a case which presented on July 27, 1914, with an acute abscess of the upper cuspid. (See Figure 417.) After the acute symptoms had subsided, the dead pulp was removed. The root was filled and the second radiograph (Figure 418) was taken on August 6. Others were taken at intervals of five or six weeks, and show the gradual building in of the bone. (See Figures 419 to 422.)

Treatment of the More Severe Cases.

From the above description of the treatment of alveolar abscess it would seem to be very simple and effective, and this is really true of the larger percentage of cases. But there are cases presenting which are much more difficult of management; cases which seem slow in the formation of pus, so that there are a number of days of excessive pain accompanied with swelling and increasing fever. In many of these a number of teeth in the neighborhood will be tender to pressure and it is sometimes difficult to locate the tooth which is the cause of the abscess. Cases occur in which the swelling of the tissues may be extensive and may be distressing and even dangerous by its interference with other functions; as for example, those swellings of the floor of the mouth or neck which interfere with deglutition and respiration; and those about the angle of the lower jaw which,

together with the spasm of the muscles attached to the ramus, make it impossible for the patient to open the mouth. There seems to be no treatment which will cut short the course of some of these cases.

Relief of pain and general symptoms.

Hot fomentations. In these a further effort to relieve the pain and general symptoms should be made. The portion of the face involved may be wrapped in a pack wrung out of hot water. A piece of flannel or coarse cloth may be dipped into the hot water and laid on the face. A heavy towel or rubber cloth laid over this will prevent the very rapid cooling of the pack. This should be kept as hot as the patient can bear for fifteen minutes or more, and then omitted for a time, and repeated as often as seems desirable. I should condemn the use of poultices of any kind applied on the face. These will soften the tissues and favor the exit of pus on the face, which is especially to be avoided. A rule should be never to allow pus to make its own opening on the skin. Whenever such an opening is imminent, a cut should be made to discharge the pus. This is for the reason that any opening made in the skin by suppuration will heal with an ugly scar, while a cut made with the knife will heal almost without a scar.

Saline cathartic. Hot foot-bath. A large dose of a saline cathartic should be a part of the regular treatment of such cases, and this should be repeated whenever necessary to keep the bowels very free until after good drainage of the abscess has been established. Together with this, the patient should have a hot foot-bath, which should be continued for fully fifteen minutes, previous to retiring each night.

Anodynes. Aspirin may be given to relieve the pain, when it is not very severe. In some of the cases in which the pain is the principal factor, not including the development of high fever or other systemic conditions of importance, I have used some form of opiate to tide the patient over this very distressing stage of the affection. In this I have been especially impressed with the usefulness of svapnia, usually in one grain doses by the mouth, because its ameliorating effect will extend over a longer period than that of the other opiates. It is not, however, so directly effective in relieving pain as some of the other forms, but it will keep the patient in a drowsy state for twenty-four hours, if the doses are properly adjusted to the individual. This has seemed to me to give relief, without otherwise influencing the

Fig. 427.

Fig. 427. Case of an alveolar abscess from the upper right cuspid which was discharging near the inner canthus of the eye. This case was referred to Dr. Gilmer's clinic at Northwestern University Dental School. See description of case in the text.

Fig. 428.

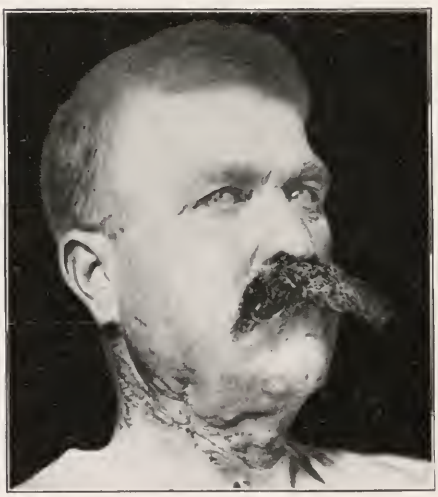

Fig. 429.

Figs. 428 and 429. Case of alveolar abscess from a lower third molar. This patient was operated upon by Dr. Carl E. Black. See description in the text. Figure 428 was taken immediately after the operation; Fig. 429 after the wound had healed.

progress of the cases. When more prompt relief is desired, ⅛ or ¼ gr. of morphine may be given hypodermically.

Drainage. When in the progress of these cases the pus has finally been discharged, the same treatment to maintain good drainage should be used as in the other cases. This effort at cleanliness should be vigorous from first to last. In all cases there should be a close watch for the decomposition of pus, and sufficiently good drainage should be maintained to prevent it. By these several means, the more severe cases can generally be brought to favorable termination.

Burrowing of pus. Sometimes it will be found that the pus in rather small amounts has been burrowing in this direction or that, the directions being variable, so that no one detailed description would give a correct idea of them. These are to be looked for continually in the treatment of such cases, and if signs of such burrowing should be found, they should at once be investigated, and the incision extended as may be necessary for drainage. I have sometimes followed such burrowings down upon the neck, or into other regions in tracing out the directions taken by the pus. A knowledge of the anatomy and of the fact that pus is liable to become entangled in the fascia of muscles, or in the muscular tissue itself, and follow its fibers, is always something of a guide as to the directions the pus may take, but a description that will cover the cases which may present seems impracticable. The finding of these will depend most on the acuteness of the observer.

I recall one case in which a patient presented with a tremendous swelling of tissues of the floor of the mouth and of the neck from the left sterno-cleido-mastoid muscle around to the position of the mental foramen on the right side. The tongue was also much swollen. There was discomfort in swallowing, the patient was suffering severely, had a high fever and could not open the mouth more than about three-eighths of an inch at the incisors. No area of fluctuation could be found by palpation, there was nothing in the history of the case to indicate the tooth which was responsible for the abscess, and it was out of the question to make a satisfactory examination of the teeth. Although it was not expected that pus would be obtained, the skin was frozen with a spray of ethyl chlorid and an incision was made at about the center of the swelling. The cut was made through the skin, about one and one-half inches below the lower border of the bone at the position of the left mental foramen, the direction of the blade being such that the point reached the bone near its lower

border. The point was then scraped along the bone, in order to certainly cut through the periosteum for possibly an inch. The swelling was so great that a little more than all of the blade of an ordinary scalpel was within the tissues. There was considerable hemorrhage, but no pus. The incision was packed to prevent it from closing. Anodynes were given, hot fomentations applied, a saline cathartic was ordered, also a hot foot-bath before retiring.

The next day there was practically no change in the patient's condition, except that the difficulty in swallowing was increased, and the patient was alarmed because there was some slight interference with respiration. No pus could be palpated. The knife was inserted into the opening made the previous day and, after reaching the bone, was carried farther along it in every direction, but no pus was found. Anodynes, hot fomentations, a saline and foot-bath were again ordered. On the third day conditions were practically the same. The patient was very weak and very much alarmed. Still no pus could be palpated. The knife was again inserted into the previous opening, but on reaching the bone was directed to the lower border, under it and a short distance upward on the lingual side of the bone. This incision was successful in reaching the pus and possibly half a teaspoonful was discharged. The wound was irrigated and packed and the patient's recovery was rapid.

In this case, as was afterward learned, the abscess was caused by a dead pulp in the lower second molar. The pus had penetrated the bone on the lingual side, below the attachment of the mylo-hyoid muscle, and had evidently followed along the inner surface of the bone, keeping below the attachment of the muscle, ripping up the periosteum without penetrating it. It was out of all question to locate the pus by palpation, as the mouth could not be opened sufficiently to admit a finger between the teeth, and there was so much swelling that nothing could be learned by an external examination; it was only possible to locate the position of the lower border of the bone by noting it on the opposite side. This was an unusual case in the route which the pus followed, and it was only after very extensive searching that it was found. It is probable that had this patient been taken to the hospital and anesthetized, the mouth could have been opened and the pus located and discharged more promptly, but conditions were not favorable for so doing.

A very interesting case presented at our school clinic several years ago, and was under Dr. Gilmer's care in the department

of Oral Surgery. A young man came in to have his teeth examined, and it was noticed that there was a sore on the right side of his nose almost level with the inner canthus of the eye. A little pus was discharging from this sore. The patient stated that the discharge had occurred at intervals for more than a year and all efforts to cure it had failed. In examining the mouth a sinus was discovered above the right cuspid root. In exploring this, the Examiner passed a sharp steel probe into the sinus in the mouth and it came in contact with the end of the cuspid root, about which there was a cavity within the bone. Further exploration with a silver probe revealed a sinus which terminated with the opening on the side of the nose. The case was then referred to the Oral Surgery Clinic and Dr. Gilmer found a necrosis of the right nasal bone. With the removal of the bone and the extraction of the tooth, the case made a prompt recovery. Figure 427 shows the case, with a probe passed through the sinus and protruding from the opening on the side of the nose.

The following is a report of an unusual case of alveolar abscess, taken from the case records of Dr. Carl E. Black, of Jacksonville, Illinois:

"G. B., male; age 48; farmer; married; father of two children; circumstances moderate, home surroundings pleasant; hard worker; no bad habits; general physical condition had always been excellent; family history good. Urine showed a trace of albumen and numerous granular casts.

"The last of January, 1910, a dentist extracted the lower right third molar on account of pain and soreness about the tooth. About March 1, patient began to have soreness beneath the chin and a little later swelling on right side of lower part of face. After several days he consulted a physician and was referred back to the dentist, who told the patient that he could find no cause in the mouth for the swelling and pain. Patient then consulted another dentist, who thought some part of the molar tooth was still in the jaw and was the cause of the trouble. Patient then went to another physician, who gave him a prescription for his throat and applied antiphlogistine locally as a poultice for about two weeks. The condition grew worse, and the patient consulted another physician who at once decided that the case demanded surgical intervention and sent him to Passavant Hospital and to my care.

"When I first examined the case, March 31, 1910, there was a tense, painful, fluctuating swelling involving the right side of the face and extending from the mastoid process to beyond the

median line of the chin and down to the clavicle, from its attachment to the sternum to its middle. There was a constant purulent discharge from the mouth, pouring out from the cavity where the molar tooth had been extracted.

"The patient was given a general anesthetic (ether). After shaving the face and neck an incision was begun at the angle of the jaw and extended to the middle of the clavicle. This exposed a large abscess cavity below the fascia, or rather a series of abscess cavities connected together by sinuses. A second incision was made below the mastoid and a third in the median line just under the chin. All pockets were opened and broken-down tissue removed and all incisions connected by drainage tubes, as shown in Figure 428. The main cavity below the angle of the jaw was packed wide open with gauze. One cavity was below the sterno-clido-mastoid muscle. No attempt was made to close any part of the incision. The discharge through the mouth at once ceased and the wounds healed rapidly. Patient left the hospital on April 23, practically well, but with an ugly scar on the face as shown in Figure 429.

"I did not see this patient again until October 5, 1910. Examination at this time showed that the pain in the right arm at the time of the acute process was not simply a referred pain but that as sequela of the infection he had had a brachial neuritis resulting in permanently diminished sensation in the palmer surfaces of the fingers of the right hand. The muscles between the thumb and the first metacarpal and between the metacarpals of the little and ring fingers of the right hand were much atrophied, and the power of the hand diminished fully one-half for purposes of manual labor."

As one reads the report of this case, as of many others of the more severe cases, it seems evident that a more painstaking and thorough examination leading to a proper early diagnosis would indicate the treatment necessary to cut them short.

Prophylaxis as Applied to Alveolar Abscess.

Since alveolar abscess is a sequela of the death of the pulp, all that has been said relative to the prevention of hyperemia and inflammation of the pulp, and of the treatment of hyperemia, is the best prophylaxis against alveolar abscess. There should be added the necessity of careful observation and watchfulness, directed especially to those conditions which precede abscess development.

If a tooth should have a hyperemia, the dentist should make

a record of the condition and should examine the tooth at subsequent periods to ascertain if the pulp is still alive. If the hyperemia is severe the tooth should be watched until it has subsided, giving special appointments for this purpose, if necessary. If a tooth has occasionally been sore, it should be examined to see whether or not the pulp has died, and if it has, treatment should be undertaken at once. Delay will probably mean that the patient will present at some future time with an acute alveolar abscess. The most thorough investigation should be made of every indication that a pulp may be dead or dying.

On board a steamer crossing the Atlantic, the ship's physician asked me to see a patient. The gentleman had come aboard seemingly well, but had developed a pericementitis, which was running rapidly into a suppurative period, assuring an acute alveolar abscess within a few hours. The pain was already very severe and, as there was no possibility of finding instruments for the rational treatment of this, I advised him to lose the tooth. There were several pair of forceps on board, and I removed the tooth. I should not have thought of removing this tooth if the case had been in my own office. This gentleman had visited his dentist a few days before leaving home, and it had been discovered that the pulp of this tooth was dead, but the dentist told him that he would let it go until the patient returned from this trip, which would be within a few weeks. This was the result.

CHRONIC ALVEOLAR ABSCESS.

ILLUSTRATIONS: FIGURES 430-460.

Etiology.

THE termination of the acute form of alveolar abscess with the chronic form immediately following, and persisting, is one of the marked characteristics of the disease. My observations indicate that, unless the dead pulp is promptly removed after the subsidence of the acute stage, the chronic form follows in as many as 98 per cent of cases; in other words, for an alveolar abscess to get well without passing into the chronic form, unless the pulp is removed, is the exception to the very general rule. However, the soreness usually disappears after a short time, so that the tooth may again be used in mastication without discomfort.

While the development of the chronic alveolar abscess is generally a sequel of an acute abscess, the acute abscess should not be considered so much the direct cause of the chronic abscess as is the dead pulp. If the acute abscess receives prompt and thorough treatment, and the dead pulp is removed from the tooth and a good root filling made, a chronic abscess does not develop as the rule. There are three principal causes of the development of the chronic abscess: (1) Infection through the root canal; from a dead pulp which may remain after an acute abscess, or which may cause a chronic abscess without the intervention of the acute form; or following imperfect technic in connection with the treatment of the canal; (2) Destruction of the apical fibers of the peridental membrane by an acute abscess; (3) Destruction of the apical fibers of the peridental membrane by strong antiseptics placed in the root canal.

In tests of the power of the bite with the gnathodynamometer in a large number of cases, most patients will stop short of the pressure upon these teeth which will be exerted upon the other teeth, showing such teeth to be lame, although they may not have noticed this difference in the ordinary use of the teeth. This lameness shown by the gnathodynamometer will usually be found to some degree in all cases in which chronic alveolar abscess has healed permanently. In practically all such, the

FIG. 430.

FIG. 431.

FIG. 432.

FIGS. 430, 431, 432. Lower jaw of a Flat-Head Indian from Columbia River, Oregon, showing destruction of bone by alveolar abscesses from both first molars. In Figure 430, the open pulp chambers of both of these teeth may be seen and in Figures 431 and 432 the cavities in the bone about the distal roots are typical of the injury which occurs.

FIG. 433.

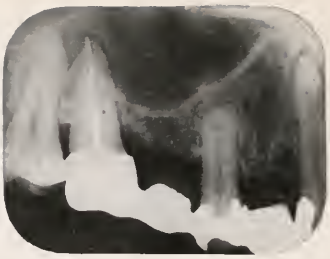

FIG. 434.

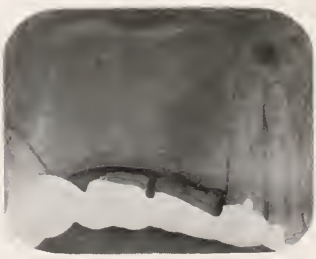

FIG. 435.

FIG. 436.

FIG. 437.

FIGS. 433 TO 437. Radiographs which emphasize the necessity of careful diagnosis.

FIGS. 433, 434. Radiographs of a case of alveolar abscess. Figure 433 shows the condition when the patient first presented. It looks as though both bicuspids might be involved. However, the pulp of the second bicuspid was tested and found to be alive. The first bicuspid was extracted. Figure 434 shows the condition a year later. The pulp of the second bicuspid remained vital.

FIG. 435. In this radiograph the mental foramen is shown close to the roots of the lower bicuspids. There were four blind abscesses in this patient's mouth, and at first glance one might mistake the mental foramen for an abscess. In view of the fact that the second bicuspid is supporting one end of a bridge and the root canal has not been filled, an abscess from this tooth might be expected.

FIGS. 436, 437. The radiograph shown in Figure 436 was taken for a rhinologist in an examination of the maxillary sinus, and the first molar was ordered extracted, with the belief that the dark area above it was an alveolar abscess. The pulps were found to be alive in the second bicuspid and first and second molars, and another radiograph was ordered taken at a different angle. In this one the "abscess" does not appear.

peridental membrane about the apex of the root is never as strong as before. The gnathodynamometer is shown in Figure 102.

Pathological changes.

Destruction of the periapical tissues. It must be said of the fibers of the peridental membrane attached to the apex of the root that they show a very persistent vitality. In the beginning of acute alveolar abscess, it would seem that these fibers would be destroyed in much greater proportion than the facts indicate; generally, they are not destroyed by the inflammation in the acute form of alveolar abscesses. They seem to take little part in this process. The bone is quickly absorbed; the pus seems to seek the cancellous portion of the bone which is near by and the pus cavity is formed in this bone. Very little pus remains among the fibers of the peridental membrane, which have been in a degree loosened from their attachment. It is largely for the reason that the abscessed cavity is transferred to the cancellous portion of the bone surrounding the end of the root that these fibers escape destruction. I think this will be seen by every one who examines many radiographs showing pus cavities.

In those cases in which the abscess cavity is not well drained during the acute stage, there is, naturally, more danger of destruction of the apical fibers of the peridental membrane.

If dead pulp remains in tooth. If a dead pulp remains in a tooth, subsequent to an acute abscess, a chronic abscess will be maintained by the discharge of infectious material through the apical foramen. If, in such cases, no serious injury has occurred to the peridental membrane up to the time when the case presents, the proper treatment and filling of the root canal should effect a cure.

If periapical tissues destroyed by acute abscess. If the acute abscess should have destroyed the apical fibers of the peridental membrane, or, if from the continuous subsequent infection from the root canal, these fibers are destroyed, the abscess never can heal so long as that area of detachment remains.

If periapical tissues destroyed by drugs. If drugs, such as phenol and some preparations including formalin, which will destroy tissue, are permitted to come in contact with the apical fibers of the peridental membrane, this tissue will be permanently destroyed and the abscess can never heal.

Detachments permanent and maintain chronicity. Therefore, after such a detachment occurs, the case continues as a

chronic abscess, even though the pulp may be removed and a good root filling is made. Then the abscess may only be cured, either by cutting off the end of the root, or extracting the tooth, for a reattachment to such an area of cementum is impossible, as we have seen in our studies of the detachments occurring in diseases beginning at the gingival line. It is for this reason that such abscesses are liable to discharge continuously and resist all efforts for a cure.

The denuded portion of the cementum in this case acts precisely in the same way that a spicula of necrosed bone acts in any other part of the body. A recently formed chronic abscess will usually heal and remain well after the cleaning of the root canals, and the filling of the apex of the root. If, however, such an abscess has persisted for several years, the chances for a cure are diminished in proportion to the time, for some destruction of the peridental membrane over the end of the root practically always occurs sooner or later.

The maintenance of the chronicity of such cases by the denuded cementum, which is necessarily pus-soaked, is no new principle. It has been recognized from far back in the history of the development of pathology, that a spicula of pus-soaked necrotic bone remaining in the tissues will keep up suppuration indefinitely. Mr. Lister, in his struggle to render a true account of the influence of micro-organisms in wounds, makes this statement in a paper he presented to the Royal Society of London in 1867.* It will be noted that this statement was made before the establishment of the present known facts of the production of suppuration by micro-organisms. It runs thus:

"Further, it shows that the mere contact of a foreign body does not of itself stimulate granulations to suppurate; whereas the presence of decomposing organic matter does. These truths are even more strikingly exemplified by the fact that I have elsewhere recorded (Lancet, March 23, 1867) that a piece of dead bone free from decomposition may not only fail to induce the granulations around it to suppurate, but may actually be absorbed by them; whereas, a bit of dead bone soaked with putrid pus infallibly induces suppuration in its vicinity."

This applies with equal force to cementum which has lost its soft tissue attachment and has become pus-soaked. This pus-soaked cementum can not be loosened from its attachment to the root by absorption, for neither the cementum, nor the dentin,

* Epoch-Making Contributions to Medicine, Surgery and the Allied Sciences, by C. N. B. Camac, A.B., M.D., p. 13.

have any power in or of themselves to produce an absorption that would loosen such a bit of necrosed tisssue, because they have no circulation of red blood, nor living elements, that can perform such an absorption. Therefore, it becomes a permanent interference with the healing process.

Classification. The several forms of chronic alveolar abscess may be classified as follows: (1) Chronic alveolar abscess with a sinus; (2) chronic alveolar abscess discharging through the root canal; (3) blind alveolar abscess, from which there is no route of discharge, the contents of the cavity being absorbed by the tissues; (4) chronic alveolar abscess with intermittent or periodical discharge.

Chronic abscess with sinus. In the most common form with a sinus, there is a direct route for the discharge of pus, extending from a cavity in the bone about the apex of the root, through the alveolar process and soft tissues to the surface of the gum in a position immediately over the root of the tooth which has caused the abscess. Less frequently, as will be described later, the point of discharge may be considerably remote, either within the mouth or upon the face or neck. (See Figures 404, 414, 415 and 416.)

Chronic abscess discharging through root canal. Those cases in which the discharge is through the root canal are not different from those having a sinus, except that the root canal has offered the route of least resistance. These are liable at any time to take on a more acute form by the plugging of the root canal with food debris. (See Figure 402.)

Blind abscess. The term blind alveolar abscess seems appropriate as descriptive of the form having no route of discharge. This term is widely known and used by the profession, and I know of no English term which describes it better. A very limited number of abscesses of this type are amenable to treatment. Cysts are more likely to be formed in this than in any other class of cases. It is probable that a very large proportion of these have more or less characteristic elements of cyst formation. Figures 438 to 441 are radiographs of cases showing blind abscesses.

Chronic abscess with intermittent discharge. This form is practically a combination of the other forms. An abscess with a sinus may change to the blind form by the closing of the sinus; it may then discharge again. These changes may occur frequently. Figure 442 is an illustration of an abscess of this type.

Variations in position of sinus openings. While the open-

ing of the sinus will usually be immediately over the root of the diseased tooth, this is not always the case. Sometimes, especially when the opening is on the face, the sinus may be long and tortuous. I recall a case in which the opening was through the gum exactly over the root of a lower left cuspid. This tooth gave a positive response to heat, as did the other front teeth, and as both bicuspids and the first and second molars had been extracted, the dentist had not been able to determine the cause of the abscess. A probe followed this sinus distally to the root of the third molar. Occasionally the discharge will be at the gingival line, the peridental membrane having been detached, so as to form a sinus alongside the root.

Some of the cases are more obscure. Formerly, or in the early history of my own practice, I saw many neglected cases in which sores had persisted for a long time upon the face, or even well down upon the neck, from this cause. The pus from these abscesses tends to gravitate downward. It often becomes entangled under the fascia or along the course of the fibers of muscles, and follows these. In many cases it becomes entangled in the platysma muscle and follows along the direction of its fibers. It is not uncommon for such entangled pus to break out upon the skin over the prominence of the sterno-cleido-mastoid muscle and for a time discharge in that position; or it may pass over the sterno-cleido-mastoid muscle, and still following the fibers of the platysma muscle, discharge at the position of the clavicle. Occasionally the pus will pass to the inner side of the clavicle and enter the pleural cavity with fatal result.

I have seen cases in which there was a scar upon the gum where the abscess had originally discharged; another scar under the angle of the jaw, another along the line of the sterno-cleido-mastoid muscle, and a fresh sinus opening on the clavicle. In this way they leave their record, having broken out here and there along their course, discharging at one place for a time, and then gravitating further and forming a new place of discharge. Such cases are now very rare. The change is the result of the spread of information in regard to such cases, so that people find relief very much earlier now than formerly.

In all of the cases in the lower jaw, the pus takes a course in a degree similar to that which I have related. Occasionally it finds an exit toward the lingual, but it is rare for it to burrow far. From the lower incisors, the discharge may pass through the lower border of the bone and make its exit below the chin. (See Figures 415 and 416.)

FIG. 438.

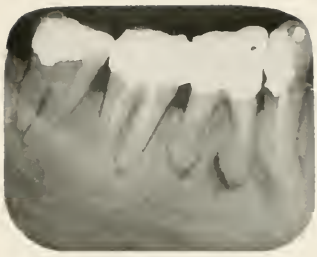

FIG. 439.

FIG. 440.

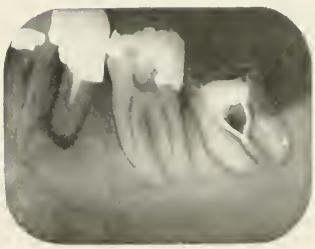

FIG. 441.

FIGS. 438 TO 441. Four radiographs showing blind abscesses. Figures 438 and 439 both show blind abscesses from both mesial and distal roots of lower first molars. In Figure 440 the abscess is from the root of the upper bicuspid. In Figures 438 and 440 there seems to have been no attempt at root fillings. In Figure 441 there is a blind abscess from the lower second bicuspid, also an abscess from the lower second molar, and holes were found through both roots near the bifurcation. Wires were put through these and the radiograph was taken to convince the patient that the tooth should be extracted.

Fig. 442.

Fig. 442. Photographs of plaster model of a case of chronic abscess with intermittent discharge, from mesial root of lower first molar. A sharp steel probe was passed into the sinus and it was found that there was an extensive destruction of bone about the root, similar to that shown in Figure 431. The tooth was extracted.

From an upper tooth, if the discharge is on the face, it is usually somewhere along the malar bone or under the malar prominence. (See Figure 411.) A very few cases have come under my observation in which the pus has become entangled in the fibers of the masseter muscle and has followed down to its attachment in the lower jaw. From that position it has usually made its exit about the angle of the jaw; a few possibly becoming entangled from that point in the fibers of the platysma muscle, or other muscles and going farther in a downward direction. These cases are very uncommon.

It happens also that abscesses from the roots of the upper molars, and bicuspids as well, often discharge into the maxillary sinus, producing suppuration of its mucous membrane. (See Figure 408.) The bone covering the apices of the roots and forming the floor of the maxillary sinus is often very thin, so that pus may readily penetrate it. There is little doubt but a large percentage of cases of suppuration of this sinus are caused by alveolar abscesses. A specialist in diseases of the nose and throat stated to me recently that in his opinion fully eighty-five per cent of maxillary sinus infections resulted from alveolar abscesses. In other cases, particularly from the incisors, the discharge may be into the nasal cavity, and perhaps be difficult of discovery. (See Figure 407.)

The discharge from an abscess from the root of an incisor tooth may take a backward course and follow between the mucous membrane and the bone, and make its exit at the junction of the hard and soft palates. I am persuaded that this discharge may occur upon the upper side of the soft palate, but it has been discovered usually upon the lower side.

Such discharges of pus from chronic alveolar abscesses occur in exceptional cases, so that the number seen by any one man is not often very large. I have had the opportunity to see unusual numbers, so that my experience has been very much wider than that of the majority of practitioners of dentistry. In my practice, before I began teaching, I was in continuous contact with physicians and surgeons and was called in consultation in many of these cases by a tolerably wide range of medical men; and in the dental school clinic, I have seen most of the unusual cases which have presented from among eight thousand to thirteen thousand patients per year.

Deposition of subperiosteal bone. In cases of blind abscess, there often is more or less enlargement by the deposition of subperiosteal bone upon the surface, while the bone is

hollowed out within. This enlargement occurs in the same way that subperiosteal bone — the involucrum — is occasionally built in cases of necrosis. In cases of abscess this newly formed bone is usually so thin that it will give a little if pressure is made with the finger on the overlying gum tissue. Many of these are overlooked until there is quite an enlargement from the cause above mentioned.

If a stiff, sharp, steel probe is placed upon such an enlargement of the bone about the apex of the root and considerable pressure applied, it may go through into a cavity, developing the fact that a blind abscess of some size exists. In some of these the sharp steel probe will go through into the cavity very easily, while in others it will require heavy pressure to push it through, owing to the different thicknesses of the bone. This bone is usually quite dense, and a thick shell of it is hard to penetrate.

Deposits of serumal calculus. Whatever portion of the cementum is denuded of its peridental membrane may receive a deposit of serumal calculus. Such a deposit occurs under conditions similar to those under which serumal calculus is deposited on the cementum of pus pockets resulting from inflammations of the gingivæ. The denuded cementum absorbs products of the suppurative process, and is more or less irritating to the adjacent soft tissue, from which serum is exuded. This carries its proportion of calco-globulin whenever there is any in the body fluids, and a deposit occurs on the root. (See Figures 451 to 454.)

Diagnosis.

The local symptoms of chronic alveolar abscess may be few, yet a diagnosis is usually made with little difficulty. There may be a history of an acute alveolar abscess, which should always lead one to suspect that a chronic abscess may be present.

Pain. There may be no complaint of pain in these cases; in fact, many persons doubtless have chronic alveolar abscesses for years without being conscious of their presence. The patient may complain of a feeling of fulness in the region of the tooth, or of a dull aching within the jaw or face at intervals. There may be pain almost anywhere on the same side as the diseased tooth, which may be associated with the inflammation about the tooth.

Tenderness of tooth. The patient may have noticed that the tooth was lame, or tender to heavy pressure. The tooth may

have been periodically sore in biting upon it. In a good many cases the tooth will do its usual work without attracting attention, but whenever something harder than usual comes upon it in chewing, it is lame. The tooth may never have been noticeably sore or lame, but the patient may notice a difference in sensation when the diseased tooth and several others in the neighborhood are tapped with an instrument, or if lateral pressure is made with the fingers; or there may be no sign whatever of lameness. The lameness is likely to be in proportion to the amount of tissue destroyed about the apex.

Absorption of bone and looseness of tooth. If the abscess is of long standing and some bone has been destroyed about the apex of the root, the tooth may be loose. This is because the fan-shaped apical fibers, which ordinarily hold the apex in its normal position, have been destroyed. By a digital examination the outline of the root may be made out, it being plain that the alveolar process is missing.

Pulp of tooth dead or removed. There will, as a rule, be no response to thermal or electrical tests for the vitality of the pulp of such a tooth. Such tests, however, are not decisive in themselves. Some teeth, especially if much abraded, give no response, although the pulp may be vital. In teeth having more than one root, the portion of the pulp in one canal may be dead, and that in another canal may retain its vitality for some time and there might be a response. There may be several teeth in the neighborhood from which pulps have been removed, so that the one causing the abscess can not be differentiated from the others. The test should be made, however, as an aid in diagnosis.

A tooth containing a dead pulp is likely to be darker in color than the other teeth; in fact, any tooth from which the pulp has been removed, may be discolored. In those cases in which the pulps of undecayed teeth have died from hyperemia or traumatism, the discoloration may be the only sign of a blind abscess.

Discharge of pus. Pus may be discharged more or less continuously from a sinus about the apex of the root of the tooth. This may continue through half a lifetime, or more. I have observed teeth with chronic alveolar abscesses, from which the pus had been discharging for many years, although the teeth had been useful every day in mastication. The quantity of pus discharged is very small; often not more than a drop every few days. As has been mentioned, the discharge may be intermittent, the sinus opening and closing again when about so much pus has accumulated. In this way it swells and breaks every

few days, or a week or two may pass between these discharges, during which time the abscess seems well and there is nothing but a scar at the point at which it breaks.

In some cases in which there is a discharge every few days, the tissue closing between times, the mucous membrane will be pouched out and will form an excessive granulation through which the pus escapes. If this is removed, it will be reformed. If the sinus opening is on the face or neck, the discharge may be either continuous or intermittent, as in cases discharging upon the gum.

As has already been mentioned, there may be no sinus and the pus may escape through the root canal and pulp chamber into the mouth. In these cases also, the discharge may be continuous or intermittent. If the cavity in the tooth is in such a position that it may be packed with food, the escape of the pus may be interfered with and an acute abscess may develop.

A number of cases presenting as blind abscesses are evidently abscesses from which pus had escaped through a sinus for a time and later the sinus closed, yet the disease had continued about the apex of the root, gradually destroying more of the periapical tissues.

In a number of cases, particularly those in which the original injury is caused by the placing of powerful antiseptics in the root canal, there will be a slight discharge of clear serum through the root canal into the pulp chamber. I have seen a discharge of this character in a few cases in which I, personally, know that it was not caused by antiseptics, but such cases have been rare. In the majority of these cases, we must conclude that the injury by the drug has preceded the infection. In some cases the teeth may become persistently painful, as well as sore to the touch, and may remain so after the root canal treatment has been completed. Again the denuded cementum keeps the apical tissues in a constant state of irritation.

I remember a case in which an oculist sent me a patient for examination, with the statement that there was an inflammation of the eyes which looked something like trachoma but evidently was not. He wanted to know if there was any condition about the mouth which might affect the eyes. I discovered a chronic abscess over the root of an upper central incisor which evidently discharged intermittently. The tissues were swollen at the time and it was apparent that the pus would be discharged within twenty-four hours. In reply to a question, the patient said it was occasionally painful and she would break the abscess with

her finger, pressing out a little pus, which would give relief. The abscess shown in Figure 442 is one of this type.

Suspecting that she might carry the infection to the eyes on her finger, I immediately undertook the treatment of the abscess. I cleaned the root canal and within a couple of days the pus discharge ceased. I then sent her back to the oculist, telling him what I had found and the treatment employed. He subsequently reported that the eyes were practically well and required no further treatment.

Extent to which cementum is denuded. Examination with steel probe. The most important condition to be determined is the extent to which the cementum has been denuded, for this indicates the treatment to be employed. It is usually not difficult to discover that a chronic alveolar abscess exists; it is sometimes difficult and generally very important to learn the exact condition of the tissues involved. The sharp, stiff, steel probe, first suggested for this purpose by Dr. Thomas L. Gilmer, is by far the most important single means to be employed for this purpose. (See Figure 465.) The probe should be passed into the sinus, and it will usually come directly in contact with the denuded end of the root. If there is much of a cavity in the bone, the contour of the root can be made out; also the amount of bone destroyed may be determined. This simple examination is often sufficient to a full diagnosis and to indicate the proper treatment. A soft probe with a blunt end is of little service as compared with the sharp steel probe. Figures 431 and 432 give a good showing of the conditions which may be felt with the sharp probe. It would be very easy to follow the contour of these roots, also of the cavities in the bone.

Radiographs. In most cases of chronic alveolar abscess, no matter which form, there will be sufficient destruction of bone about the apex of the root to be definitely shown by a good radiograph. The use of the radiograph is now becoming so general, and the employment of small films for use in the mouth give so much better definition of the teeth and maxillary bones, that it should be the rule of practice to have radiographs made of all cases of chronic alveolar abscess, as an aid in making a complete diagnosis. There are, of course, many cases in which the other symptoms, and particularly the findings with the sharp steel probe, will be sufficient to indicate that the tooth should be extracted. However, in all cases in which the effort is to be made to save such a tooth, a radiograph should be made.

Figures 438 to 441 and 443 to 450 are radiographs of various types of abscesses. Figures 433 to 437 are shown to illustrate some of the errors which might be made in reading radiographs.

TREATMENT OF CHRONIC ALVEOLAR ABSCESS

HISTORICAL.

In the early years of my practice of dentistry it was the habit of dentists whom I knew to extract all teeth presenting which had abscesses at their roots, the impression being that such cases were incurable. I very soon discovered for myself the means of curing many of these abscesses by cleaning the root canals, and filling the apices of the roots of the teeth. At first I filled the root canals with gold, and the treatment was limited for some years to those teeth more readily approached, as the front teeth, but gradually others were similarly treated. No instruments for this purpose could be obtained from the dealers. I had to make them myself. I succeeded in making very many of these teeth useful and healthful, so far as I could determine.

At that time I supposed that this process was original with myself. As time passed and my acquaintance with dental literature became wider, I discovered that others had done the same thing before me, but for a number of years the knowledge of such things was confined to a comparatively few dentists, and physicians and surgeons had no knowledge of it at all.

Once when I was in a neighboring city I was shown the results of a surgical operation in which a cancer was said to have been removed by cutting out a section of the lower jaw. I quickly showed that this so-called cancer was nothing more than an alveolar abscess, presenting a rather unsightly appearance upon the face, and might have been cured by extracting the tooth, or possibly by cleaning the root canals of the tooth and filling them.

I will recite one other case which occurred twenty-odd years ago. It serves to illustrate the lack of knowledge of these cases, also the disadvantage of the use of strong antiseptics. One evening, while I was at work in my laboratory, a physician, who shared his office with a dentist, both of whom I knew well, entered my office and came directly to my laboratory. He told me that they had a case which had puzzled both the dentist and himself. The patient had a sinus on the neck, a little under the

point of the chin, and they could not discover the cause. I at once asked him if they had examined the lower incisors carefully. He said they had not. I told him to look for a lower incisor which was darker than the others, and if there was such a tooth to extract it at once. Accordingly, the next day an examination was made, and such a tooth was found and extracted. On examination of its socket for necrosed bone, he found a large opening which led through the body of the bone to the site of discharge on the tissues below. An ordinary excavator could be passed through the opening from inside the mouth to the outside below the chin. Vigorous treatment was at once instituted. A little swab was wrapped on an instrument, this was moistened with phenol and passed through the opening. This was repeated every day for several weeks, but the abscess refused to heal. Finally, very bad weather came — it was in the winter; a storm prevented the patient coming in for about ten days, as she lived several miles in the country, and to the surprise of these gentlemen the abscess had healed. Had they left the case alone after extracting the tooth, there being no necrosed bone, the sinus would have closed within a few days. It could not heal while that particular treatment was continued.

When I first began the treatment of chronic abscess with a sinus, I used creasote which I injected through the apical foramen of the tooth into the abscess, when I could do so, sometimes pumping it in with cotton wrapped on a broach. In this way I could send the drug clear through, filling the abscess in pretty much all its ramifications until the drug appeared on the tissues on the outside.

After such a treatment as this, an abscess of recent formation — that is, one that had just passed from the acute to the chronic form — would almost universally get well with a single treatment.

When phenol came into use, this was substituted for the creasote and used in the same way, and was pumped through the root of the tooth until it appeared at the opening of the abscess. This was also generally successful in the class of cases stated above, one treatment being sufficient.

It was later discovered that the pumping of phenol through into the abscessed cavity until it came out upon the surface was unnecessary. If the root was thoroughly cleaned and kept so by the use of an antiseptic in the root canal, such an abscess would generally get well without other treatment. That is to say, if the reinfection through the canal was stopped, the tissues

in and of themselves took up and destroyed the micro-organisms acting to produce pus, and effected a cure in that way.

My treatment of these cases was gradually simplified, both as to the number and frequency of the treatments and the strength of the drugs used. A permanent filling was made at the apex of the root, as soon as the pus formation ceased, with the belief that nothing would be gained by further treatment through the canal; that the continuation of the abscess, if it did not heal, would be due to injuries which had occurred to the tissues about the apex of the root and which would not be benefited by root canal medication. The object was to prevent future ingress of micro-organisms through the root canal. This general plan of treatment, which will be given in detail in the following pages, has become a standardized routine procedure which may be applied to practically all of the ordinary cases which present.

Treatment.

After having made a clear diagnosis of the conditions in chronic alveolar abscess, the course of treatment should be determined. As has been mentioned, the most important thing to learn, previous to undertaking treatment, should be the extent of the detachment of the peridental membrane from the cementum about the apex of the root. If this tissue has not been seriously injured, a speedy cure may be expected. On the findings in the examination, it should be decided first of all, whether the tooth should be extracted at once, or if an effort should be made to save it. If much of the root is denuded, the effort to save the tooth should not be made unless the amputation of the denuded portion of the apex is contemplated.

Treatment of root canal. The first step in the treatment should be directed to the cleansing and subsequent filling of the root canal, as has been given under the treatment of the pulp. In this connection I will only mention here that the removal of the dead pulp and the thorough sterilization of the root canal should be sufficient to cure most of those abscesses which are not complicated by destruction of the apical fibers of the peridental membrane. If, after removing the dead pulp, a mild antiseptic dressing has remained sealed in the canal for a week, and the discharge of pus has ceased, the root should be filled without further delay. Or, if there is some improvement, but the discharge has not entirely ceased, a second treatment may be sealed in for a week. There are very few cases in which I would consider further treatment of the canal of any advantage, and I

would, as a rule, fill the root canal after the first treatment, or in occasional cases after the second treatment, whether the sinus had closed or not. I see no logical reason for further treatment of the canal. The failure of healing is due to conditions outside the root, which can not be improved by treatment of the canal. The canal should, therefore, be filled, and subsequent treatment, if necessary, directed to the periapical region through the gum and alveolar process.

When sinus does not heal. If the case does not heal subsequent to the filling of the root canal, the existing sinus should be enlarged, or a new opening through the gum and alveolar process made, in order that the end of the root may be resected. Sometimes there may be small spiculæ of dead bone in the abscess cavity, and if found, these should be removed. If these methods fail, the tooth should be extracted.

Cases of blind abscess. In cases of blind abscess, in which, on account of the constant leakage of the contents of the abscess cavity into the root canal, it is impossible to thoroughly dry the canal, so that a good root filling can be made, a dressing should be sealed in the canal and the filling of the root delayed until the abscess cavity has been drained by an opening through the gum. There will then be no difficulty in drying the canal.

In order to be able to reach the abscess cavity in the bone, novocain may be injected, and a curved incision from one-half to three-fourths of an inch long should be made through the soft tissues over the root, the convexity of the curve being toward the crown of the tooth. The tissue on the concave side of the cut should then be dissected up from the bone, and held with a small retractor or other instrument. Then, with a bibeveled drill or fissure bur in the engine, the opening through the outer plate of bone may be enlarged until there is free access to the abscess cavity within the bone. If the case is a blind abscess, the drill is used to cut away the outer plate in exactly the same way. No harm is done in cutting away a considerable amount of bone, so long as the drill is not permitted to approach too near the root. The greatest care should be taken not to injure the peridental membrane.

After making such an opening, the case should not be allowed to heal by closure of the superficial tissues, but should be kept open until the deeper parts have healed. A small strip of gauze may be introduced to keep the wound open, changing this every two or three days; less gauze being used as the wound heals.

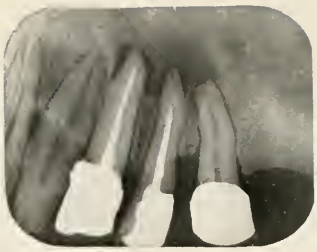

Fig. 443.

Fig. 444.

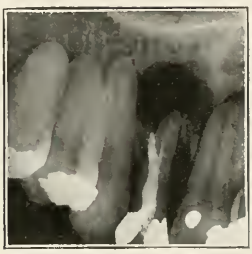

Fig. 445.

Fig. 446.

Fig. 447.

Figs. 443 to 447. Radiographs of cases of chronic alveolar abscess. In both Figures 443 and 444 the roots of three teeth are involved. Figure 445 shows a gutta-percha filling in a hole which had been drilled through the mesial side of the root of an upper lateral incisor. This appears to have been the cause of the abscess in this case. The cuspid root has been exposed by the abscess cavity. Figure 446 shows a cuspid root, with very little attachment left, supporting one end of a large bridge. This abscess evidently originated from the lateral incisor, and the condition about the cuspid root should have been discovered before the bridge was made. Figure 447 shows an abscess from an upper lateral incisor. This root was resected.

FIG. 448.

FIG. 449.

FIG. 450.

FIGS. 448, 449, 450. Three radiographs showing alveolar abscesses. In Figure 448 it appears that a crown was set on the cuspid without treating the root canal, or at least without filling it. In Figure 449 the principal destruction of bone has been between the roots of a lower molar. In Figure 450 a very good root filling is shown in an upper lateral incisor. The bone was probably destroyed before the treatment of the canal was undertaken. A radiograph at that time would have given the necessary evidence for a proper diagnosis.

Fig. 451. Fig. 452. Fig. 453.

Fig. 454.

Figs. 451, 452, 453 and 454. Deposits of serumal calculus on roots in cases of chronic alveolar abscess. The deposit on the side of the root in Figure 452 was in connection with an abscess resulting from a hole drilled through the root. Specimens from Northwestern University Dental Museum. In connection with the studies of the nature of the deposit of calculus, it is interesting to note that none of these positions had been exposed to the saliva, except possibly in Figure 452.

FIG. 455.

FIG. 456.

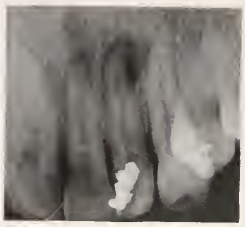

FIG. 457.

FIG. 458.

FIG. 459.

FIG. 460.

FIGS. 455 TO 460. Cases of root resection in treatment of chronic alveolar abscess. In each instance the radiographs were taken at least a year after the operation. Figures 455, 456, 459 and 460 are from cases operated by Dr. Thomas L. Gilmer. Figures 457 and 458 are before and after operation, patient of Dr. Arthur D. Black. It will be noticed that in each case bone has been built into the space formerly occupied by the end of the root.

This in brief constitutes the routine treatment of the chronic alveolar abscess. A treatment which is so uniformly successful, when success is possible, that it may be followed with the expectation of good results, in practically all cases in which the apex of the root is not denuded.

Practice in vogue should be discontinued. The practice in vogue for so many years of treating these cases, first with this drug and then with that, without having made careful examination of the conditions, should be entirely discontinued. The extent to which the peridental membrane has been detached should be determined first. In cases in which there is any reasonable doubt as to the existing conditions, generally one simple test treatment should be made to learn whether or not the case is complicated by the denudation of the cementum. It is useless to follow this up with a varied assortment of drugs with the hope of finding something that will cure. Strong antiseptics may inhibit pus formation, but they at the same time so lower the vitality of the tissues that the discharge from such cases will be renewed later. One who makes records of such cases will find this to be true. The regular routine treatment of alveolar abscess here given has proven dependable throughout many years of practice in which cases have been followed by careful records.

The basis for the change of this treatment lies in this fact: Whenever a strong antiseptic is used in any of these cavities, the phagocytes, which follow up and actively destroy micro-organisms that may be in the tissues, withdraw and cease their activity in combating the infection. In fact, the vitality of all of the tissue with which the antiseptic comes in contact is reduced. For this reason, the cure of the abscess is much slower than that which occurs with the simpler treatment above detailed.

Records of a large number of recently formed abscesses show that very few failed to heal when caustics were not used, whereas in cases in which these were passed freely through the apex of the root, many did not heal. Subsequently an area of detachment about the apex of the root was demonstrable either by the probe before extracting, or by examination of the root after extraction.

These discoveries compelled me to oppose the passing of the stronger antiseptics through the apical foramen in the treatment of roots. If this is safeguarded, almost any of the milder antiseptics may be used for the purpose of maintaining asepsis in the root canals during the treatment of the case. I think the

general tendency of late years, among the men who think most closely, has been to use the milder form of treatment. In a pretty widespread experiment in the clinic of Northwestern University Dental School, the prohibition of the use of strong antiseptics in abscessed cavities has shown a very marked decrease in the number of cases of sore teeth, and of abscesses that failed to heal. This subject is more fully discussed in the consideration of the use of antiseptics. It still stands, however, as expressed above, that the older the abscess, the greater will be the proportion which fail to heal because of the detachment of the membrane from some portion of the apex of the root.

Resection of roots. In those cases in which the cementum at the end of the root is denuded, the tooth may often be saved and the abscess cured by cutting off the end of the root, or, to use the term suggested by Dr. Thomas L. Gilmer, by *resecting* the root. This operation should be confined to the upper incisors, cuspids, buccal roots of first bicuspids, and buccal roots of the upper molars. Resection of other roots is contraindicated by the greater depth of bone which must be penetrated, making the operation more difficult, and the results have generally been unsatisfactory, even when a good operation has been performed.

Technic. The technic of the operation is simple. Under local anesthesia an incision is made through the gum as previously described. The bibeveled drill is then used to cut away sufficient bone to give access to the apex of the root. Except in cases of blind abscess, it will generally be necessary to do very little cutting in the bone, as the labial or buccal plate will usually have been destroyed already. When the root is exposed it may be cut off by drilling a hole through the root and then cutting laterally in both directions from this hole with a fissure bur. The fissure bur quickly fills with the cuttings, and is objectionable on this account for drilling the hole. There is no clogging of a bibeveled drill. After the apex of the root is removed, the end of the remaining portion should be made smooth and slightly rounded.

In cases in which only a very little of the apex of the root is denuded, a large fissure bur may be used to trim off the end, without removing a definite piece. In each case, all that is required is to remove the denuded portion, and leave the remaining end smooth. This should be followed by irrigation to remove the debris and cleanse the cavity, packing sometimes being employed to permit healing from the deepest part, as previously described.

Possibilities of healing. One might think of the conditions presenting as to the healing of the tissues over such a root end, as being identical with those of a pus pocket resulting from a detachment of the peridental membrane beginning at the gingival line, in case all of the pus-soaked cementum were removed from a denuded area, but the two are not quite parallel. In the case of the resected root, it is a matter of a few days at most until the root end is entirely enclosed within the tissues and completely shut off from the fluids of the mouth, so that a reinfection is very much less likely to occur.

There are three possibilities of healing: (1) In the absence of infection the tissues may attach themselves to the root end; (2) the cementoblasts from the sides of the root may gradually build new cementum over the end; (3) the tissues may simply heal over the root end, without being attached to it, the same as they might heal around a bullet. It is my opinion that this last is what really happens in the large majority of cases.

It should be stated that a percentage of these cases do not do well. The root end causes sufficient irritation to keep up a slight discharge and the opening through the gum does not entirely heal. Or, if it does heal, the irritation causes the constant outpouring of slight quantities of serum and a pocket remains about the root end. In either case the elements for the reproduction of the chronic abscess are present, and sooner or later, it is formed. All cases in which roots are resected should be carefully watched, and within a year or so a radiograph should be taken to learn the condition, even though the tissues look to be all right. The cases which do not succeed can be cured by extraction. Figures 455 to 460 are reproductions of radiographs in cases in which roots were resected. These were taken from one to three years after the operation.

Amputation of molar roots. When a chronic abscess involves but one root of a molar, the diseased root only may sometimes be amputated. This operation is more often indicated for lingual roots of upper first molars, and less often for either root of a lower first molar. The technic of the operation has already been given in connection with the treatment of suppurative pericementitis. In cases of abscess, more or less of the peridental membrane remains attached between the abscess and the gingival line, so that the position of the bifurcation of the roots, their general contour, etc., is not so easily determined as in cases of suppurative pericementitis. (See Figures 290 to 293.)

NECROSIS OF THE MAXILLÆ

ILLUSTRATIONS: FIGURES 461-463.

NECROSIS of bone is defined as death of bone *en masse*. This occurs under widely different conditions, and in any part of the body in which there is bony tissue. An inflammation involves the periosteum covering the bone, and may extend to the bony tissue itself, establishing an osteitis. Or the inflammation may begin within the bone, as in the case of an osteomyelitis in a long bone, or as alveolar abscess in the maxillary bones, and extend to the periosteum later. During this inflammation an exudate is thrown out which becomes coagulated, and renders the central portion of the swelling harder than the surrounding tissues, which are swollen.

In the harder central portion of this area, there is stasis of the circulation; that is, the blood does not circulate in this particular portion of the inflamed area. Whenever this stasis is widespread, and long continued, and especially when it involves bone, there is likely to be death of the part of bone that is involved, because of the lack of aërated blood. It can not be thrown off immediately, like the sloughing of parts under severe inflammation and stasis in soft tissue, but remains attached to the living bone for a time. Such amelioration of conditions or of the inflammatory processes must occur as will permit the activity of the healthy bone immediately in conjunction with that which is dead. When this has occurred, a process of absorption is set up in the healthy bone close around the dead portion, by which it is finally loosened. The dead piece thus exfoliated is called a sequestrum. (See Figures 461 and 462.) Then the dead portion of the bone may be removed, sometimes in pieces, and sometimes complete in a single mass. After the complete removal of this dead portion, there is usually no hindrance to the healing process.

In most cases there is fairly complete restoration of the bone removed. In a case in which teeth are lost with the alveolar process, the alveolar process, as such, is never reformed, and often this makes quite a deformity in the mouth where large pieces of bone and teeth are lost from necrosis.

ETIOLOGY.

Necrosis of the maxillary bones frequently occurs as a sequel to the death of a pulp and the formation of an alveolar abscess; in fact, this is by far the most frequent cause. (See Figure 461.) It also occurs as a result of injury, such as fist-blows, falls, kicks of animals, etc., in which the bone may be fractured. Occasional cases of necrosis of the maxillary bones occur from arsenical poisoning, the arsenic having been used for devitalization of the tooth's pulp. Most such cases result from the placing of arsenic in a tooth, the roots of which are not fully formed, the apical ends being so large that the pulp does not die of strangulation, and the poison involves the tissues outside the end of the root. Cases also occur from arsenic insecurely sealed in teeth, some of the arsenic escaping and coming in contact with adjacent tissues and subsequently with the bone. Formerly in regions in which persons were employed in the handling of phosphorus, as in match factories, there were many cases of necrosis of the maxillary bones, due to this poison. Most such factories now engage the services of dentists to care for the mouths of their employees, and have thus reduced the number of cases to a very few.

Syphilis should also be mentioned as a cause of necrosis of the maxillary bones. The palatal portions of the maxillary bones and the palate bones are almost as frequently involved in syphilitic necrosis as are the nasal bones; in fact, most cases of necrosis occurring in the palate are syphilitic. In persons having syphilis, necrosis is more apt to occur in connection with alveolar abscess than in nonsyphilitic persons.

SYMPTOMS.

The symptoms of necrosis may be those of an acute alveolar abscess, plus the finding of necrosed bone. Therefore, severe pain and swelling, with high fever and rapid pulse, frequently mark the acute stage. In the more typical cases of necrosis of the maxillary bones, symptoms usually appear which distinguish the case as one of necrosis, without an examination of the bone. The discharge of pus is persistent, it frequently wells up about the necks of teeth in the area of bone involved. The pus is of a thick creamy consistency, and has a very foul odor, which is, of itself, almost sufficient for a diagnosis. The teeth in the area may become very loose and often fall out, or may be removed with the fingers.

If a sharp, stiff, steel probe be passed into a sinus or other

opening to the bone which is necrosed, the bone will be found to be hard and rough, often being honeycombed. The examination with this probe alone is sufficient to easily make a differential diagnosis between necrosis and chronic osteitis, the latter being very soft.

In the rise and progress of necrosis, there is much pain and swelling, as the general rule; yet I have seen cases in which the pain was not more than would be called a dull pain and the swelling was not great. Inflammations involving bone are generally more painful than inflammations involving soft tissue. After the death of the bone and the beginning of the subsidence of the inflammatory process, there is not much pain, providing good drainage is maintained. Of course, in all of these cases there is more or less inflammation and suppuration during the entire time from the death of the bone until its final removal.

In those cases in which the periosteum is held away from the bone for a time, the osteoblasts may build a layer of new bone in the new position of the periosteum. This bone is called an involucrum. It usually forms a thin shell, which will give a little on pressure. This serves to partially enclose the dead bone and it may be necessary in the treatment to break away some of this newly formed bone. (See Figure 463.) This addition of subperiosteal bone frequently occurs elsewhere in connection with disease of the bone, or where an abscess exists because of the bridging over and enclosing of pieces of necrosed bone. It not infrequently happens that the bones of the leg are very much enlarged by building on of subperiosteal bone over the deceased parts, thus strengthening the bone. When a portion of the lower jaw is cut away by disease, leaving the remaining portion rather weak, I have seen a plate of bone built out in the floor of the mouth reaching almost to the center, impeding the movements of the tongue. After the diseased bone had been separated and later replaced with new bone, this plate of bone which had grown out to strengthen the weakened part was removed by absorption. Generally these buildings of subperiosteal bone, which seem to be called out for the purpose of strengthening the weak places, will be absorbed after a time.

Treatment of necrosis.

The treatment of necrosis should be: First, the establishment and maintenance of good drainage; second, nonsurgical interference, so far as the bone is concerned, until the necrosed bone has been separated from the healthy bone; third, the care of

the patient's general health in the matter of diet, exercise, fresh air, etc.

It should be remembered that the loosening of the sequestrum is a physiological process and can be carried on only by the tissue activities in the neighborhood. This not only requires normal activity of the tissues, but also requires time, and this will be long or short, as the tissues are more or less active. Local medication can not hurry it, and will generally hinder it. It would be an entirely wrong practice to make any attempt to remove dead bone before it had been separated, because it is impossible to tell where the line of demarcation will be established, and to cut beyond into the healthy bone exposes it to the products of suppuration and decomposition, which are always present wherever there is necrosed bone. Such an operation may result not only in the loss of more bone by necrosis, but exposes the patient to the danger of a general septicemia.

Secure good drainage. The most essential thing in the treatment of necrosis is the establishment and maintenance of good drainage. Incisions should be made either inside the mouth or outside, or both, to give very free drainage. It is usually necessary to maintain drainage for a number of weeks, and some form of drainage tube or packing may be required for this purpose. Fenestrated rubber tubing may be used in the more extensive cases, or packing of gutta-percha tissue or gauze in the majority.

Cleanliness. What has already been said regarding irrigation for alveolar abscess applies to cases of necrosis. The discharge must be unobstructed and the wound should be kept as clean as may be done. If saprophitic decompositions of pus occur, it is an indication that the drainage and cleanliness are insufficient. Nothing is of greater importance to the early separation of the dead bone, and to the patient's general physical condition, than the maintenance of cleanliness. Antiseptics should not be used for this purpose, for the reason that antiseptics strong enough to counteract the activities of the microorganisms will also materially limit the activity of the tissues of the neighborhood, and do harm in this way. This treatment will reduce the fever and the swelling and thus keep the patient reasonably comfortable. Cleanliness should be maintained until the necrosed portion of the bone has loosened sufficiently to be removed. Sometimes this will be a very tedious process, requiring several weeks.

Extract loose teeth. Whenever teeth in the area are very

loose and pus is discharging about their necks, they should be extracted. During the period of irrigation, frequent examinations should be made to find pieces of bone which may have separated and each piece should be promptly removed. Oftentimes the necrosed bone will come away in a number of pieces at different times, and each piece removed reduces the inflammation and the discharge of pus. A sequestrum will often be loose, without being freely movable. A stiff, sharp instrument, such as a chisel, may be held firmly against the necrosed bone, and its mobility tested. If it has only very slight motion, so that its removal might be difficult, a few more days may be allowed, when it will usually be found to have more motion.

Cathartics and anodynes. During the acute stage, the same general treatment may be employed as in cases of acute alveolar abscess. Saline cathartics should be given, a hot foot-bath before retiring, and anodynes if necessary.

Cases of necrosis, in which the patient's general physical condition is much reduced, are best cared for in the hospital, although the majority do not require hospital service. However, the general health should be looked after in all cases, and it is sometimes desirable to call a physician in consultation for the purpose. A soft diet, moderate exercise and plenty of fresh air should be prescribed.

Removal of sequestra. In the removal of large sequestra, it may be necessary to either enlarge the opening through the soft tissues, or to break the dead bone into several small pieces to facilitate its removal. In cases in which an involucrum has formed, which serves to partially enclose the dead bone, it is usually necessary to break away at least a part of the newly formed bone, both to make the removal of the sequestrum easier, and to permit the soft tissues to close in and thus advance the healing of the wound. If this shell of bone is left as formed, it serves to maintain a cavity in the tissues for a time.

With the removal of the last piece of necrosed bone, the case will generally heal rapidly. A very small piece of dead bone may, in some instances, be sufficient to keep up a suppuration out of all proportion to the extent of the dead tissue. The treatment after the removal of the sequestrum will depend much upon the conditions in the case. It is generally best, if the sequestrum has been large, and particularly if the cavity is very deep, to pack with gauze every other day for a week or two. The packing with gauze may be employed, or not, according to the positions and relations of the parts.

FIG. 461.

FIG. 461. Necrosis. A lower first molar and a large sequestrum removed by Dr. Thomas L. Gilmer. The exposure of the pulp chamber may be seen in the right-hand illustration. Specimen from Northwestern University Dental Museum.

Fig. 462.

Fig. 462. Necrosis. Two views of a large sequestrum from the lower jaw, removed by Dr. Thomas L. Gilmer at the Oral Surgery Clinic, Northwestern University Dental School. Specimen from Northwestern University Dental Museum.

I wish to accentuate the necessity for removing every piece of necrosed bone, no matter how small, by relating this case.

A man was brought to me with a sore in the tissues below the body of the bone of the lower jaw. It was stated that this had been suppurating for eight years, and that several physicians and surgeons had endeavored to cure it by widening the opening in the jaw and curetting. I looked the case over carefully, and noticed that the first lower molar on that side was missing. The patient stated that after some years of trouble, a dentist had removed the tooth, saying that the abscess would get well, but it did not. I noticed in examining it that the full space of the first molar still remained between the second bicuspid and second molar. This aroused my suspicion of something wrong in the space formerly occupied by this first molar.

I passed a soft silver probe into the sinus, and found an opening into the bone through which the probe penetrated easily until it met an obstruction at about the position of the inferior dental canal. By bending the end of the probe a little, I finally succeeded in passing it farther into an opening under the position of the first molar, which had been removed. By several efforts I succeeded in pushing the probe forward, and it came to an obstruction which, when the distance was measured, seemed to be only a little under the tissues covering the surface of the bone in the former position of the first molar.

I made a crucial incision in the gum and laid aside all of the central portion of it with a blunt instrument. Then placing a small, sharp chisel against the bone, I directed my assistant to strike it with the mallet. At the first blow the instrument went through into a cavity. I broke off the bone sufficiently to get a good entrance, and found there a spiculum of necrosed bone, which formerly was the septum between the roots of the first molar which had been removed.

The failure to look for this and get it out when the tooth was removed, had caused the continuance of the sinus upon the face for so many years.

The discharge of pus from this abscess ceased within four days, the tissues healed, and the case remained well. This patient was so enraptured over this success in the treatment, that he practically never came to town afterward without running up to say "Thank you."

One of the worst cases of necrosis of the maxillary bones which have come to my attention was that of a man of about thirty-five who had for years been in the habit of trimming his

fingernails with his incisor teeth. He could start at one side and by a series of bites trim an even piece off the end of a nail, almost as smoothly as this could be done with a pair of scissors. One day he called on his dentist, Dr. W. B. Young, of Jacksonville, Ill., complaining that his upper incisor teeth were loose. On examination, Dr. Young found these teeth very loose, and the alveolar process necrosed; pus was being discharged at the necks of all four incisors. There was also swelling of the tissues farther back on both sides of the mouth. He extracted the four incisors, and noticed a foreign substance alongside the root of one of the centrals. This proved to be a cutting from a fingernail, which had evidently slipped up under the gingiva as it was bitten off. This apparently was the cause of the infection, which spread rapidly, and eventually involved all of the alveolar portion of both superior maxillary bones. Pus penetrated both antra and was also discharging about the necks of all of the remaining teeth. Dr. Arthur D. Black was called to see the case, and he found it necessary to remove all of the remaining upper teeth. Several sequestra came away with the teeth, including parts of the floor of both antra. During the next few weeks additional sequestra were removed. The case finally made a good recovery.

Prophylaxis against necrosis.

In the protection of patients from necrosis of bone, emphasis should be placed upon the statement that the large majority of these cases in the maxillary bones occur as a result of alveolar abscess in which pus is confined for a time between the bone and the periosteum, having parted the periosteum from the bone. (See Figures 405, 406, 409 and 413.) If the inflammation is running high and particularly if considerable fever is occurring, the danger of necrosis of bone is decidedly increased, and there should be no hesitation or delay in applying the treatment that has been indicated; that is, the full, free discharge of the pus.

This case occurred in my practice a number of years ago. A patient presented with a dead pulp in an upper lateral incisor. The tooth was sore. After placing a dam and sterilizing the field of operation, I opened the pulp chamber, and with the utmost care, cleaned the root canals, and sealed in a treatment with gutta-percha. I impressed the patient with the necessity of returning promptly if the case became worse after this treatment, telling her that there might be serious consequences from delay. Such caution has been my usual custom in the primary

treatment of cases similar to this one. This was on Saturday afternoon, and she did not return to her appointment the next week. I was uneasy, and after a few days, not hearing from her, I wrote a note, asking her to report to me. She replied that the tooth had become exceedingly sore, her face was much swollen, and that her physician said she had erysipelas, which he was treating. I again wrote her to see me as soon as she was able. About two weeks after the day on which I removed the dead pulp, she came in with her face still swollen. I then found that an alveolar abscess had developed at the root of this tooth and had formed a broad, flat swelling, extending distally along the jaw, and that the bone was necrosed for a corresponding distance, but was not yet ready for exfoliation. When exfoliation did occur, the sequestrum included the lateral incisor, cuspid and the two bicuspids, and opened into the maxillary sinus. This undoubtedly would all have been avoided by prompt treatment of the abscess.

In this case the physician did not recognize the real cause of the swelling, but finding the face very much reddened, he came to the conclusion that it was erysipelas, and the abscess received no treatment.

I recall a case of a young man of twenty-two, who presented with an abscess at the root of a central incisor. The pus had burrowed along the bone, raising the periosteum from it, and was discharging between the gingivæ and the teeth throughout that side of the mouth to the third molar, and necrosis of bone had become established. I extracted the teeth from the central incisor to the third molar, and removed the necrosed bone, including the buccal portion of the alveolar process, and a portion of the floor of the maxillary sinus. By breaking away some of the lingual portion of the alveolar process, which had not necrosed, I brought the tissues across the opening, stitched them together, and the case made a very good recovery. In a number of cases in which necrosis has involved the floor of the maxillary sinus, I have drawn the tissues together over the opening after the bone has been removed, and generally there has been no future inflammation within the sinus. I sent the young man to another dentist to have a plate made when the case was ready for it, and a piece was so adjusted that he could wear it with comfort; but still it was a fearful loss, caused by neglect of the proper treatment before the case came to me.

CHRONIC OSTEITIS OF THE MAXILLÆ

ILLUSTRATIONS: FIGURES 464-465.

CHRONIC OSTEITIS* may be defined as death of bone, cell by cell. Chronic osteitis differs so widely from necrosis of bone that it must be considered an entirely separate disease. It is a condition of inflammation and disintegration which is progressive in its character, involving the bone in absorption and separation of its parts, in which we have a soft mass that may enclose more or less small hard particles of necrosed bone.

The condition of the progress is such that the bone is disintegrated cell by cell, instead of being destroyed *en masse* as in necrosis. Chronic osteitis shows a very decided disposition to continuous slow progress, attacking and softening the bone to which it makes approach, often hollowing out the cancellous portions of large areas of bony tissue. This portion of the bone is seemingly preferred by this process, although upon occasion it will burrow through the hardest bone. The disease is generally marked by what we would term a chronic condition in its whole progress.

Etiology.

The cause of chronic osteitis is a peculiar form of infection, probably symbiotic†. The spread of this infection to the adjacent parts is progressive, but slow. In the mouth, chronic osteitis

* In dental literature, this condition has been commonly referred to as caries of bone. The word caries was formerly applied in surgery to a cellular destruction of bone caused by tuberculosis, actinomycosis or syphilis. The condition of the maxillary bones here described is similar, but seems not to result from the causes mentioned.

† The growth of two or more micro-organisms together, producing different results from the growth of any one alone.

Among the micro-organisms in the mouth there are several symbiotic combinations with accidental organisms, and they are somewhat frequent. The best example is perhaps that which produces green stain, which is seen oftenest on children's teeth, consequently I will use it as an illustration. If a pure culture of caries fungus is planted in a Petri dish, spreading it on about one-half inch of surface, and after this has grown about two days, one of the molds, penicillium glaucum or penicillium nigre, is planted about one-half inch or so from the caries growth in four places around it, this will grow very much more quickly than caries fungus, and its mycelium will run in every direction in the sub-stratum. Wherever the mycelium crosses the caries fungus a cloud will appear, and as more of them cross, the cloud will increase in depth. No such cloud, however, will occur if the plants are kept separate. The cloud results from the combination of these growths.

A child, who has some obstruction which interferes with normal breathing, will not breathe well when sleeping unless the mouth is open. The gingival portion of the

Fig. 463.

Fig. 463. A boy who had had an acute alveolar abscess from a lower first molar which resulted in an extensive necrosis. After the dead bone was separated, a piece nearly two inches long remained within the tissues for a number of months, pus discharging through a sinus on the neck below the lower border of the bone. The periosteum, which had been lifted from the outer plate of the bone by the abscess, formed an involucrum, part of which had to be cut away in removing the sequestrum. This picture was taken before the operation. The apparent swelling of the left side of the face is due to the new bone, which held the soft tissues in this position. There was really very little inflammation of the overlying tissues at this time. Patient of Dr. Arthur D. Black.

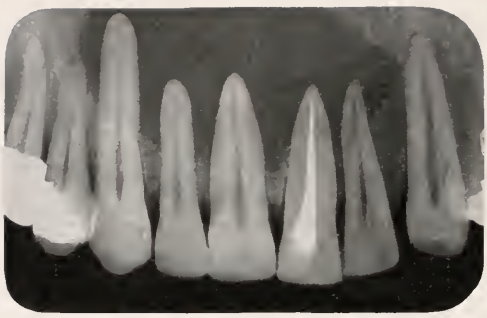

Fig. 464.

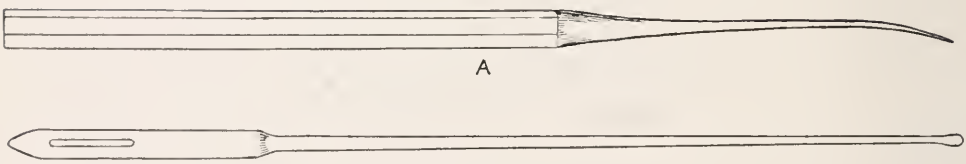

Fig. 465.

Fig. 464. Radiograph of an extensive case of chronic osteitis, patient of Dr. Joseph Eisenstaedt. In this case there was a rim of alveolar process which held the teeth firmly in place, while practically all of both superior maxillary bones from midlength of the roots to and including the floor of the nose, as far backward as a line drawn across from the right cuspid to the left second bicuspid, had been destroyed. The radiograph does not show so large a cavity, as the full thickness of the bone was not destroyed over the entire area.

Fig. 465. A, The sharp, stiff steel probe first suggested by Dr. Thomas L. Gilmer. With such a probe one can, with a little experience, easily differentiate the enamel of an impacted tooth, the root of a tooth, necrosed bone, or the softened bone in chronic osteitis. This probe should be used to explore every sinus about the mouth. B, A soft, blunt end silver probe for following tortuous sinuses.

occurs most generally as a sequel of blind alveolar abscess, although, it may occur following other forms of chronic alveolar abscess.

It is a rather curious fact that chronic osteitis seldom occurs in the lower jaw, and most frequently in the incisor region in the upper jaw. The number of cases which have occurred about the roots of upper lateral incisors have each year attracted attention at the Oral Surgery Clinic in Northwestern University Dental School. This should be a particular warning in our care of this tooth. It should be remembered that the pulp of the upper lateral incisor is more liable to hyperemia and inflammation, on account of the small size of the tooth, and on account of the fact that the pulp is larger in proportion to the size of the tooth than in the upper central incisor. Therefore, the pulp of the lateral will be involved earlier by caries than that of the central because there is much less dentin to be penetrated. It is doubtless often exposed by caries before the root is fully formed. Likewise, it is more liable to be exposed in cavity preparation, and is more likely to die from thermal shock. The lateral incisor is often late in its development, and the pulp is destroyed and the root filled before the apical foramen has been sufficiently reduced in size. When it is necessary to remove the pulp from this tooth, errors in technic are liable to occur on account of the small size of the canal and the frequency with which the end of the root is crooked. All of these things play a part in causing the large number of abscesses from upper lateral incisors and greater care should be exercised to prevent them.

Symptoms.

There is practically no pain and very slight, if any, swelling in cases of chronic osteitis. The temperature will seldom exceed one degree above normal. The subjective symptoms are so slight that cases will often run for years without the patient being conscious of anything wrong. There may be a little discoloration of the overlying gum tissue. A close examination may reveal one or possibly several very minute sinuses, about the openings of which there may be little rings of granulation tissue.

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labial surfaces of the upper incisors will be kept moist by the saliva, which has a bountiful supply of caries fungus growing in it. In this, these molds are liable to grow during the night, and cause a little bit of color. This, occurring night after night, results in the formation of the green stain on the front teeth.

This case illustrates what is known as symbiosis — two organisms which happen to grow together, and which produce a result which neither will produce when growing alone.

The openings may be so small that they will be discovered most readily by making pressure with a finger on the gum and noting the discharge through them.

The discharge from cases of chronic osteitis is usually very slight in amount, and of a pale yellow or straw color. Unless contaminated with saprophytic organisms which produce decompositions, there is little or no odor. The pain is slight, but if there is a considerable area of bone involved, there is a systemic influence that is not expressed strongly by fever, but an indifferent depression, which is probably caused by the absorption of material from the infected area.

In Dr. Thomas L. Gilmer's book of lectures on Oral Surgery, prepared for the students of Northwestern University Dental School, he describes a case in which a man nearly seventy years of age presented with a small sinus over an upper left lateral incisor, and in operating on the case, it was found that most of the maxillary bone on that side had been destroyed by this disease. It was necessary to remove all of the teeth from the central incisor to the third molar, and the floor of the maxillary sinus had been destroyed.

Figure 464 is from radiographs of an extensive case involving most of the bone of the anterior portion of the palate.

If a small, stiff, sharp, steel probe is passed into a sinus it will usually pass into a cavity within the bone, and the end of the root of the tooth will be felt within this cavity. The probe may be passed up and down and partly around the root to determine the extent to which it has been denuded. If the end of the probe is pressed against the softened bone, it will easily penetrate it. The feeling is about the same as if the probe were pressed into a piece of unvulcanized rubber. This will definitely complete the diagnosis. Such an examination is usually painless.

Whenever there is uncertainty as to the extent of the affected area, a radiograph should be taken. One will occasionally be surprised to find by the radiograph that the condition has involved much more bone than was expected and may have denuded parts of the roots of several teeth.

Treatment.

The treatment of chronic osteitis is surgical, and should be radical. The area should be opened freely, and every particle of the softened bone removed until good, sound bone forms all of the walls of the cavity. This removal is accomplished usually and for the most part by spoon-shaped curettes, or large burs in

the engine. Then the cavity should be irrigated to remove the debris.

Generally when all of the softened bone is removed, the case makes a good recovery. In connection with the removal of the softened bone, it is necessary to either cut off the end of the root which projects into the cavity, or extract the tooth. If the end of the root is to be cut off, the root should be filled first. In the more extensive cases, in which the roots of a number of teeth are involved, these teeth must receive similar treatment. I should say that the larger the number of teeth involved, the less success may be expected in saving them by resecting the roots. It has usually been my practice to attempt to save teeth by resection in cases in which the area of bone involved was small and included but one tooth. When several teeth were involved, I have generally extracted them.

THE EPITHELIAL CELLS OF THE PERIDENTAL MEMBRANE IN RELATION TO INFLAMMATIONS AND CYST FORMATION.

ILLUSTRATIONS: FIGURES 466-476.

IN discussing the histological structure of the peridental membrane, attention was called to the chains and clusters of epithelial cells which are found lying close to the cementum, and reference was made to the possible functions of these cells.

Certain views have been occasionally expressed in our literature as to the role which these cells play in the pathology of chronic suppurative pericementitis and cyst formation. Some writers have considered them a predisposing factor in pericemental disease, contending that these epithelial cells are derived from the cells which float away in the breaking up of the enamel organs, and that as such they are in a condition of decadence. Others regard them as normal to the tissue and having the special function of limiting infections by encystment, thus classifying them as unusually active and vigorous.

It is not very difficult for one who will spend the time to follow a class of animals, or several classes of animals of different ages, through the period of scattering of the enamel organ epithelial cells to the time of their complete disappearance by absorption. Those who undertake this should have previously become well acquainted with the histological examination of the development of the teeth and of the peridental membranes. As a result of such an investigation, one will come to the conclusion that the enamel organ cells are in nowise related to the epithelial strings in the peridental membrane. In following the epithelial strings of the peridental membrane from a young animal to an aged animal, they become less abundant as the peridental membrane becomes thinner and the alveolar process hugs more closely about the roots of the teeth, but they are still found. Whatever may be the function of these cells, they are normal to the location, because they are always present in both man and the higher animals. These cells must, as a whole, be regarded

as glandular. I know of no other term to apply to them, although they have no ducts and in many respects are out of form as glands.

When I first came upon these cells and had cut a number of sections in different directions in order to get a good idea of them, I was much puzzled to know what they were, and I called them lymphatics in my first writing of them in 1886. I afterward corrected this error. I know of no other place in man or animal where epithelial cells are placed in the relation to each other as are these epithelial cells in the peridental membrane. But that they are normal to the peridental membrane is shown by the fact that they are always present.

There are long rows of these cells in the peridental membrane, reaching from the body of the gingiva, along the side of the root toward the apex, and these are placed frequently about the root. There are branches from one to the other of these rows, and there are loops which extend out from near the cementum among the fibers of the peridental membrane. (See Figures 112 to 116.) By cutting a number of sections of the peridental membrane, parallel with the long axis of the tooth and gradually approaching the root, first from one direction and then from another; as, for example, a first series approaching the labial surface, a second series approaching the mesial surface, a third the lingual, and a fourth the distal, we will get an idea of the arrangement of these cells. From observations of such sections of a number of teeth I have made a drawing to illustrate the location of these cells in relation to the root. (See Figure 466.) The figure is compounded, and has never been actually seen, but it expresses my idea of the distribution of these cells. Figures 112 and 114 also show something of the same arrangement.

In my study of these tissues I found one case in which these epithelial cells were especially invaded by micro-organisms, but I do not think that this apparent following of the epithelial strings by micro-organisms marks anything like their decadence, or failure in vigor. It only shows that there are certain micro-organisms which invade them in preference to other tissues, probably for some chemotactic reason that we do not yet understand.

The fact is that the membrane is invaded very often in long lines, not reaching widely around the root, but progressing directly toward the apex of the root; the area invaded being

very deep, but narrow. This is a factor in the pathology of the peridental membrane of great importance, and while I have not felt very free to express the opinion that these lines of cells were followed by the invading organisms, I have not been able to rid myself of that thought.

It is not my intention to follow out microbic invasions especially, but I may here express the belief that the microbic invasion is secondary to such local injuries to the gingivæ as I have described, and not to some micro-organism which is essentially peculiar to this disease. It seems probable that almost any pus-forming micro-organism would readily establish itself in such an area of inflamed tissue.

The evidence has swung away from the idea that disease of this membrane is caused by systemic conditions, to the opposite view that the so-called rheumatic and many other inflammatory conditions arise from foci of infection in the mouth or elsewhere. The relation of mouth foci to these conditions will be considered under a separate heading.

Studies by German histo-pathologists.

German investigators have recently been in a controversy over this group of cellular elements of the peridental membrane. I have a list of twenty-two articles written by sixteen different observers, most of whom seem to have made fairly complete personal laboratory studies of the histology, and especially of the participation of this system of cellular elements in pathological conditions.

This discussion has taken a very wide range. It involves studies of the characters of the cells, their derivation, their function, and their action in pathological conditions. This controversy must result in a much more accurate knowledge of the peridental membrane, both in the normal condition and in its pathological changes.

Such studies in histo-pathology should be made in perfectly fresh tissue; it should be placed in the fixative solution while the cellular elements are alive, for these live a short time after the death of the animal or of the person. In this study we must use human material.

In the Monatzschrift fuer Zahnheilkunde, for November, 1912, Professor Dr. Th. Dependorf, of Leipzig, published prac-

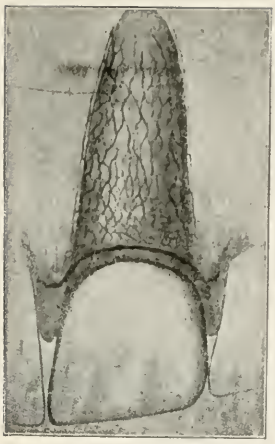

FIG. 466.

FIG. 467.

FIG. 466. Drawing made to illustrate the position of the epithelial strings in the peridental membrane. See description in text, page 493.

FIG. 467. Beginning cyst formation showing central cavity lined by epithelium. In the center of the masses of epithelium which line the cavity one sees the enlarged cells, granular and vacuolar. Many cells seem to be devoid of nuclei; the nuclei of others stain poorly. Specimen prepared in author's laboratory by Dr. H. A. Potts. Photomicrograph by Dr. F. B. Noyes.

Fig. 468.

Fig. 469.

Fig. 468. "Section through a part of an epithelial granuloma, showing band-like strands of epithelial cells among fatty degenerated and isolated granulation tissue. The arrangement simulates an atypical epithelial development. In the center is an epithelial mass (epithelglocke) which is beginning to be separated from the granulation tissue." Reproduction of illustration and translation of description, from article by Dr. Th. Dependerf, referred to in text.

Fig. 469. "Slightly oblique section through the upper jaw of an adult. Cysto-granuloma with lumen which contains pus and tissue debris. Epithelium has developed into and completely surrounded the focus. To the left of the cysto-granuloma is a softened septic focus without an epithelial covering, presenting a beginning sinus which is making its way to the surface of the mucous membrane. The root to the left is not in relation with the cysto-granuloma, which belongs to the area surrounding the apex of the neighboring tooth. Reproduction of illustration and translation of description, from article by Dr. Th. Dependorf, referred to in text.

FIG. 470.

FIG. 470. Beginning cyst formation at apex of root of a pulpless tooth. One sees fibrous tissue encapsulating a mass of younger connective tissue within which is a prolification of epithelium. In the center is a clear area lined by epithelial cells, which upon the surface have become quite stratified. Within the fibrous capsule are some solid clumps of epithelial cells. Specimen prepared in author's laboratory by Dr. H. A. Potts. Photomicrograph by F. B. Noyes.

FIG. 471.

FIG. 471. Abscess attached to apex of root showing dense fibrous tissue capsule. In the center is a mass of round cells, into which fibroblasts and new blood vessels are growing from the periphery. A few giant cells are to be seen in the younger fibrous tissue. See text, page 337. Specimen prepared in author's laboratory by Dr. H. A. Potts. Photomicrograph by Dr. F. B. Noyes.

Fig. 472.

Fig. 472. Cyst formation in alveolar abscess, most of the cavity being lined by epithelial cells. There is a well defined fibrous tissue capsule. Specimen prepared in author's laboratory by Dr. H. A. Potts. Photomicrograph by Dr. F. B. Noyes.

Fig. 473.

Fig. 473. Tissue growing into apical foramen of pulpless tooth. There are many round cells, fibroblasts and new blood vessels growing inside the canal, as well as formed connective tissue. Specimen prepared in author's laboratory by Dr. H. A. Potts. Photomicrograph by Dr. F. B. Noyes.

Fig. 474.

Fig. 474. High power of cyst wall showing the epithelial lining. Specimen prepared in author's laboratory by Dr H. A. Potts. Photomicrograph by Dr. F. B. Noyes.

FIG. 475.

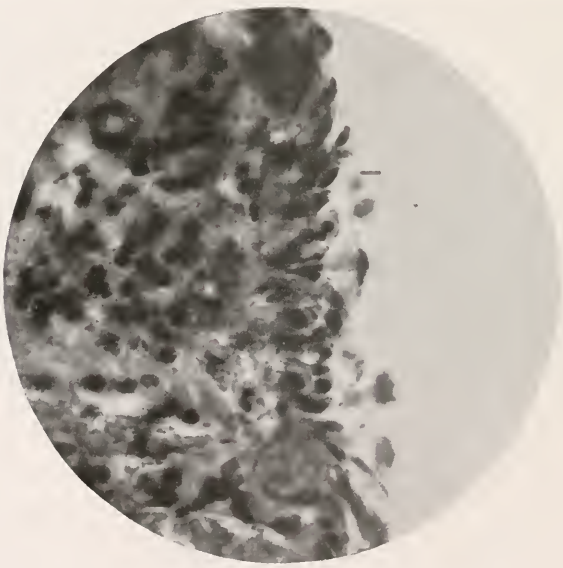

FIG. 476.

FIG. 475. Section of a wall of a large cyst, showing papillomatous growth within the cyst cavity, the whole cavity being lined by epithelial cells. The walls are fibrous and contain much round cell infiltration near the cyst cavity, while the outer part is dense fibrous tissue. Specimen prepared in author's laboratory by Dr. H. A. Potts. Photomicrograph by Dr. F. B. Noyes.

FIG. 476. High power of cyst wall showing the epithelial lining. Specimen prepared in author's laboratory by Dr. H. A. Potts. Photomicrograph by Dr. F. B. Noyes.

tically a complete review of this work up to that time.* A translation of this article was made at the time by Dr. Anna A. Oppermann.† It is not my intention in the present writing to do more than call attention to the studies which have been made and to give the principal views expressed. My recent investigations of these cells have not progressed far enough to justify a complete presentation of the subject.

The review by Dr. Dependorf showed a wide range in the views of the various writers. There is a contention that the strings of epithelial cells found in the membrane are the remains of the broken-up epithelia of the enamel organs. A number of investigators speak of these cells as epithelial remains. Others take the view that these cells are normal to the peridental membrane, and are not in any sense whatever remains from the breaking up of the epithelia of the enamel organs. Some of these observers seem to have followed this point carefully from the child at term for several years, and declare that the scattered epithelia from the enamel organs break up more and more, are absorbed and disappear completely; that the epithelial strings are normal to the peridental membrane and bear no relation to the epithelial cells scattering away from the enamel organs of either the deciduous or permanent teeth. I have made very thorough studies of these cells, and this latter view agrees perfectly with my own. This is the view given prominence by Dr. Dependorf. These cells are always present. They differ in number and in prominence in different animals, but are never absent. They are most abundant in the herbivora and omnivora; least in the carnivora.

Cyst formation.

The activities of these epithelial cells in pathological conditions are the most interesting feature of the studies referred to above. They place them in the principal role in the formation of cysts in the peridental membrane, and in the tissues of the neighborhood. There are thirty-one illustrations in the article by Dr. Dependorf, of which I reproduce two. (See Figures 468 and 469.) One shows a complete encystment of an infected area, the other a section of an encysting wall.

The word cyst is from the same derivation as our more common word cistern, a space walled off to catch and store rain-

* Zur Pathogenese der Zahnwurzelzystem. Deutsche Monatzschrift für Zahnheilkunde, 1912, p. 809.

† Northwestern Dental Journal, Vol. X, p. 73; Vol. XI, p. 9.

water. In the pathological sense, a cyst is a space which contains a fluid, walled off by a membrane. Cysts may result from injuries or disease. The limiting membrane is usually epithelial, though there are cysts with membranes of endothelium of the type of the lymphatic system. These serve to wall off portions of tissue which have been injured. In the skin and mucous membranes injuries often occur to the ducts of sebaceous or other glands, causing the retention of the secretion by the closure of the duct. The fluid contained in a cyst may vary from a thick, fatty matter, to a thin, opalescent fluid.

In order to have a cyst, it would seem that epithelium should form the limiting membrane. In the positions I have just mentioned, the epithelium of the glands, or gland ducts, is at hand for this purpose. In the formation of the cysts which are so frequent in the peridental membrane, the epithelium composing the epithelial strings and clumps of cells appears to play a principal role.

In case of an inflammatory movement, there is at once a new growth, or increased growth, in these epithelial strings. If there is in the neighborhood a group of cellular elements which are unable to maintain their vitality, they are hedged about at first by the growth of these epithelial strings. As the area of seminecrotic tissue enlarges, these epithelial strings broaden into sheets, which tend to surround and enclose it. If this movement of the tissues is complete, the area will also have become surrounded by connective tissue cells outside of the epithelium. This constitutes the formation of a cyst.

As a cyst, it may enlarge and wander in the direction of least resistance, usually gathering to itself more fluid contents. The cyst may thus assume considerable dimensions and may destroy adjacent tissues by the pressure resulting from its growth. Or, on the other hand, the cyst may show the opposite tendency, and become progressively smaller until it is obliterated.

These conditions result from infection. Chronic alveolar abscess has preceded most of those which have been studied. Many micro-organisms are enclosed within the cyst walls, and more seem for a time to be gathered in. These and the cellular elements, which also wander in with them, disappear by solution. Curiously enough, no matter how many micro-organisms may appear in the fluid of a cyst, none will grow. Everything entering the cyst, enters to its death, whether microbe, leucocyte,

or connective tissue cell. The contained fluid is sterile, yet when spilled out into the tissues it may be toxic.

From the clinical observation that the cyst is a bag containing a fluid or semifluid material within a membranous capsule, and that severe inflammation has sometimes followed the premature rupture of this membrane, surgeons have become especially careful in dissecting them out complete. If a portion of a cyst wall is left in the tissues, it will usually lead to the redevelopment of the cyst, or even of several cysts, more or less closely joined together.

The men who are now engaged in these studies are giving the formation of small cysts in the peridental membrane, especially in the apical portion, a good deal of importance in connection with the diseases of these membranes. Many roots of teeth, with chronic alveolar abscesses, will, when extracted, bring away masses of soft tissue of various sizes, attached to the cementum, at the border line of the denuded portion of the root. If this tissue is prepared for microscopic examination, many of the smaller specimens, and some of the larger ones, will be found to be cysts of this character.

During the past two years I have been studying these cysts and have had reproductions made of a number of photomicrographs of sections prepared in my laboratory, which illustrate the elements composing them. Figures 467, 470, 471 and 472 are sections through the entire circumference of the surrounding walls, showing variable amounts of epithelial elements lining the cavities, these being enclosed by connective tissue. Figures 474 and 475 are higher magnifications of sections of these cyst walls, and Figure 476 is a still higher magnification of the cells which line the cyst.

SYSTEMIC EFFECTS OF CHRONIC INFECTIONS OF THE MOUTH

THE loss of the investing tissues and final loss of the teeth constitute only the local side of the picture of the mouth infections which have been considered. There is another side that is even more important. During the progress of the diseases of the investing tissues of the teeth, whether caused by deposits of salivary calculus, or in the form of pus pockets alongside the roots, or chronic alveolar abscess, there is a continuous inflammation of low degree and almost continuous suppuration, and the pus formed usually is itself undergoing putrefactive decomposition through the growth of the saprophytic organisms.

The view that serious systemic infections occur as a result of suppurations in the mouth has been powerfully stimulated recently by a few notable papers. Articles on this subject have been a prominent feature of both the medical and dental literature of the past four years. In reviewing three dental journals and one medical journal, I found about fifty articles published during a single year.

Dr. Hunter's paper on oral sepsis.

Dr. William Hunter, a surgeon of London, published an article on Oral Sepsis* in 1911, in which he lashed the dental profession unsparingly for allowing chronic abscesses and other forms of chronic suppurations to continue in the mouth. Dr. Hunter called attention to the fact, as it had never been done before, that the foci in the mouth are in the same causal relation to arthritis, nephritis, cholecystitis, endocarditis, etc., as are infected tonsils, or chronic suppurations in any other location. Dr. Hunter was especially severe in his denunciation of the habit of placing plates over infected roots, anchoring bridges to abscessed teeth, or teeth with inflamed and suppurating gums, or placing artificial crowns on such roots. Any

* Role of Sepsis and Antisepsis in Medicine, Lancet, Jan. 14, 1911.

construction that gives unclean pieces of artificial replacement came in for his condemnation.

The following paragraphs from Dr. Hunter's article give his views of the important relation of oral sepsis to the general health:

"In my clinical experience septic infection is without exception the most prevalent infection operating in medicine, and a most important and prevalent cause and complication of many medical diseases. Its ill-effects are widespread and extend to all systems of the body. The relations between these effects and the sepsis that causes them are constantly overlooked, because the existence of the sepsis is itself overlooked. For the chief seat of that sepsis is the mouth; and the sepsis itself, when noted, is erroneously regarded as the result of various conditions of ill-health with which it is associated — not, as it really is, an important cause or complication.

"The causal connexion between the two sets of processes — the sepsis and its ill-effects — can be demonstrated by the simple expedient of removing the sepsis, and noting the striking effects which the removal has upon the existence, character and intensity of the ill-effects. The ill-effects referred to include in individual cases every one of the diseases described in the foregoing section, and regarded as essentially medical in their character — viz., the general ill-health, dirty, sallow complexions, the indigestions, the gastric and intestinal troubles, the anæmias which resist treatment; tonsillitic, pharyngeal and glandular troubles of children; the chronic rheumatisms, obscure fevers and blood poisoning, etc.

"The effects are not the same in all cases, any more than are the effects of septic infection in surgery or those of tuberculous infection in medicine. They affect sometimes one system and sometimes another, in different degrees according to the individual susceptibility, just as a chronic tuberculous infection may in one case affect the glands of the neck, in another the joints, in another the bone, in another the lung, in another the meninges, in another the peritoneum, and in another it may affect them all. . . . . .

"Sepsis in medicine therefore ranks, in my experience, as the most prevalent and potent infective disease in the body. It therefore deserves the particular attention of the whole pro-

fession as much as it has hitherto received their particular neglect. It requires this attention at the hands of every branch of the profession. . . . . .

"It is an all-important matter of sepsis and antisepsis that concerns every branch of the medical profession, and concerns very closely the public health of the community. It is not a simple matter of 'neglect of the teeth' by the patient, as is so commonly stated, but one of neglect of a great infection by the profession — a great infective disease for which the patient is not primarily responsible any more than he is responsible for the contraction of typhoid fever or tuberculosis. The condition referred to is that to which I have given the name of 'oral sepsis.'

"The title 'oral sepsis' was first introduced into medical literature in a paper entitled, 'Oral Sepsis as a Cause of Disease.' (British Medical Journal, July, 1900.) My object in seeking for a special name, and after consideration in creating this one, was to emphasize the great fact that it is not the absence of teeth but the presence of sepsis; that it is not dental defects, but septic effects; that it is not defective mastication, but the effective sepsis associated with such dental defects, or often present in conditions of gingivitis apart from such defects, that are responsible for the ill-health associated with 'bad' mouths.

"The second object was to emphasise the importance of the infection caused by staphylococcal and streptococcal organisms, as distinguished from the purely saprophytic infections in which the mouth abounds; or the temporary presence of specific organisms — e. g., typhoid, tubercle, pneumonia, etc.

"The subject of 'oral sepsis,' as I designated and defined it — namely, the septic lesions of streptococcal and staphylococcal infection found in the mouth — belongs to no one department of medicine or surgery. It is common ground on which the general doctor, physician, or surgeon; the throat, nose, ear and eye specialist; specialists in children's diseases, in stomach diseases, in blood diseases, in 'rheumatic' diseases, in fevers, in skin diseases, in nervous and mental diseases, and, lastly, the dental surgeon, all meet on terms of equal responsibility. In its earliest manifestations no special knowledge is required to deal with it; a sound grasp of the principles underlying anti-

sepsis alone is required. Unfortunately for the patient it is precisely this grasp which I grieve to say is wanting.

"But, it is urged, the condition is so common that it is impossible either to prevent it or to deal with it successfully; further, the ill-effects are few and rare compared with the wide prevalence of the condition. As I originally showed (1900), and my further experience of the last ten years, supported by that of many others, fully demonstrates, the ill-effects are both common and grave. That they are not more common is due solely to the great resisting power possessed by the mucosa of the mouth and gums.

"This matter of oral sepsis is, therefore, of urgent importance in relation to the whole multifarious and widespread group of affections — medical, surgical and dental — caused by the actual presence of toxic action of pyogenic organisms (staphylococci and streptococci)."

Dr. Billings' investigations.

In reviewing many of the articles upon this subject which have appeared during the past few years one is impressed with the fact that Dr. Frank Billings, of Chicago, and several of his associates, particularly Dr. E. C. Rosenow, have made the most painstaking and thorough studies of the relationship of local foci of infection to general systemic conditions. It seems that Dr. Hunter and many others have recognized the relationship principally as a result of clinical observation, and more especially by noting the improvement which has followed the extraction of the teeth in hundreds of cases. Dr. Billings and his associates have found the organism in the original focus in the mouth or tonsil, have found the same organism in the inflamed joint, or other secondary lesion, have cultivated this organism, injected it into animals, produced identical lesions in the animals and finally recovered the organism from the affected tissue of the animal. This chain of evidence establishes beyond question the relationship of the local focus and the systemic condition. These studies have also included preparations of the tissues involved secondarily, some of the material, as in cases of arthritis, being taken from the living subjects, other material being obtained from the secondary lesions produced in animals.

As the papers by Dr. Billings and his associates practically

cover the field, I give a list of their writings as the best series for reference.*

An interesting symposium appears in the Journal of the American Medical Association of December 5, 1914 (Vol. 63, p. 2023) to which Drs. Gilmer, Billings, C. H. Mayo, Rosenow and Craig are contributors. The work of these men, and many others, has served to establish without question the direct relationship of the mouth focus to the secondary systemic lesion.

In this symposium Dr. Billings says: "Systemic disease due to a focus of infection anywhere, is probably always hematogenous. The study of the infected tissues of experimentally inoculated animals and the infected muscles, joint tissues, lymph-nodes proximal to infected joints, nodes on tendons, etc., of patients, yield specific bacteria, and histologically there is found embolism of the small and terminal blood vessels. Local hemorrhage and endoarterial proliferation result in interstitial overgrowth, cartilaginous, osseous, vegetative and other morbid anatomical changes, dependent on the character of the tissue infected.

"Partial or complete ischemia of the tissues due to the embolism is an important factor in the production of the morbid anatomic changes. Oxhausen of Berlin has produced, in animals, aseptic osteochondritis resembling arthritis deformans, by ligating the arteries supplying the joint tissues. These principles are, I think, susceptible of proof, that a chronic alveolar

* Chronic Focal Infections and Their Etiologic Relations to Arthritis and Nephritis. Frank Billings, Archives of Internal Medicine, Vol. 9, 1912, p. 484. Also, Illinois Medical Journal, Vol. 21, 1912, p. 261.

Chronic Oral Infections. Thomas L. Gilmer, Illinois Medical Journal, Vol. 21, 1912, p. 275.

Chronic Focal Infections as a Causative Factor in Chronic Arthritis. Frank Billings, Journal of the American Medical Association, Vol. 61, 1913, p. 819.

Clinical Aspect and Medical Management of Arthritis Deformans. Frank Billings, Illinois Medical Journal, Vol. 25, 1914, p. 11.

Transmutations within the Streptococcus-Pneumococcus Group. E. C. Rosenow, The Jour. of Infectious Diseases, Vol. 14, Jan., 1914, p. 1.

The Etiology of Acute Rheumatism, Articular and Muscular. E. C. Rosenow, The Jour. of Infectious Diseases, Vol. 14, Jan., 1914, p. 61.

Lesions Produced by Various Streptococci; Endocarditis and Rheumatism. E. C. Rosenow, New York Medical Journal, February 7, 1914.

Etiology of Arthritis Deformans. E. C. Rosenow. Journal of the American Medical Association, April 11, 1914, p. 1146.

The Newer Bacteriology of Various Infections as Determined by Special Methods. E. C. Rosenow, Journal of the American Medical Association, Sept. 12, 1914, p. 903.

The Medical Management of Chronic Arthritis. Frank Billings, Illinois Medical Journal, September, 1914.

Focal Infection, Its Broader Application in the Etiology of General Disease. Frank Billings, Journal of the American Medical Association, Vol. 63, 1914, p. 2024.

Bacteriology of Cholecystitis and Its Production by Injection of Streptococci. E. C. Rosenow, Journal of the American Medical Association, Nov. 21, 1914, p. 1835.

infection, and chronic foci in other regions also, may cause systemic disease by hematogenous bacterial emboli, which infect and at the same time deprive the tissues of nourishment. Local infection of muscles, joint tissues, etc., and lessened blood-supply result in the peculiar morbid anatomy of the respective tissues.

"To investigate and manage these patients requires team work of the clinical and laboratory workers. The clinician must carefully examine the patient, exhausting every detail in the personal history. The skill of the dentist, the nose and throat specialist, the gynecologist, the genito-urinary expert and others may be necessary to locate the foci of infection. The focus must be destroyed. Tissues and exudates of foci should be carefully examined and bacterial cultures made. Vaccines of the dominant bacteria may be made for subsequent use. . . .

"Autogenous vaccines may be used in the attempt to improve the defenses of the body. In chronic arthritis, with the circulation of the infected tissues obstructed embolically, antibodies in the blood stream, even if augmented by vaccines, would have but little effect locally. . . . When the local circulation has been so improved that the tissues are flooded with blood, vaccines will be of undoubted value."

Dr. Rosenow's studies.

Dr. Rosenow calls particular attention to the differences found and the changes which occur in the organisms in the foci. He says:

"One striking thing in connection with some of the more chronic infections is that the character of the micro-organisms found in the lesion may be quite different from the character of the micro-organisms in the focus of infection at the same time. This, however, does not minimize the importance of the focus of infection in any way. The organisms found in the tissues may have undergone change. This fact should be borne in mind whenever autogenous vaccines are to be used. The poor results in some cases of arthritis, for example, following the use of autogenous vaccines prepared from the tonsils or other presumable focus, may be due to the fact that the organisms present in the focus at the particular time when the cultures were made were not like those actually infecting the tissues. And if so, the vaccine would fail to contain the proper antigen. . . . .

"My study of the effect of varying degrees of oxygen tension on the members of the streptococcus group, together with other facts, makes it likely that it is in the focus of infection that

changes in virulence occur and the different affinities for various structures are acquired. In other words, the focus of infection is to be looked on not only as the place of entrance of the bacteria, but also the place where the organisms acquire the peculiar property necessary to infect. In the light of our present knowledge the argument that infections in the mouth are so common in individuals in apparent health, does not minimize their importance. These or other foci are so common in patients suffering from arthritis, neuritis, appendicitis, ulcer of the stomach, cholecystitis, goiter, etc., and so rare in individuals who have had superb health for years, that their direct etiologic role can scarcely be questioned."

One of the most interesting features of Dr. Rosenow's work was presented in a paper entitled, Bacteriology of Vascular Infection, read before a joint meeting of the Chicago Surgical and Pathological Societies, on January 8, 1915. Dr. Rosenow reported the results of experiments on animals with pure cultures of streptococci. These were from cases of appendicitis, ulcers of the stomach, cholecystitis and arthritis in humans, and the injections were made into animals, mostly rabbits and dogs, usually into the ear or leg veins. Subsequent examination of the animals revealed the fact that the streptococci in a large majority of cases produced lesions in the animals in the same locations and of similar character to those in the person from whom the culture was obtained, for example:

Fifty-nine animals were injected with fresh cultures from cases of appendicitis, and of these 41 were found to have developed inflammations of the appendix; while 5 showed either ulcer or hemorrhage of the stomach, 1 an inflammation of the gall bladder, 17 of joints, 13 of the endocardium, 5 of the myocardium, 7 of muscles, 4 of kidneys, and 4 inflammations elsewhere in the intestines.

Seventy-nine animals were injected with fresh cultures from ulcers of the stomach, and of these 47 were found to have developed hemorrhage of the stomach or duodenum, and 50 developed ulcers, while only 2 showed inflammation of the appendix, 20 of the gall bladder, 3 of the pancreas, 10 of joints, 9 of endocardium, 5 of myocardium, 5 of kidneys, and 7 lesions in the intestines.

Twenty-seven animals were injected with fresh cultures from cases of cholecystitis, and of these 22 developed inflammations of the gall bladder, while none showed inflammation of the appendix, 9 showed hemorrhage or ulcer of the stomach, 1 of

pancreas, 5 of joints, 4 of endocardium, 1 of myocardium, 3 of muscles, 2 of kidneys, and 2 inflammations in the intestines.

Seventy-one animals were injected with fresh cultures from cases of arthritis, and of these 47 developed joint inflammations, 33 of the endocardium, 31 of the myocardium, 19 of the pericardium, 19 of muscles, and 28 of kidneys, while only 6 developed inflammation of the appendix, 16 hemorrhage and 13 ulcers of the stomach, 2 inflammations of the gall bladder, and 2 of the pancreas.

Similar experiments were conducted by injecting streptococci which had been cultivated through several generations. These caused inflammations markedly less in number, and showed much less tendency to establish themselves in the same locations in the animals as in the individuals from whom the cultures were obtained.

These experiments seem to have established beyond question the importance of the blood stream as a carrier of infection, and also the very peculiar and as yet unexplained tendency for the organisms to cause the same diseases in the animals as in the individuals from whom the cultures were obtained.

The organisms in the primary focus.

It is an interesting fact that, in all of the cases of this group, the primary foci is a suppurative lesion, while the secondary systemic condition usually is not. It must be that the pus-producing organism in the primary focus does not produce the secondary lesion, or else the morphology of the organism is materially changed. If the pus-producing organism of the original focus is not changed, then the secondary manifestations are caused by a non-pyogenic organism which accompanies or follows the pus producer into the original focus.

The systemic lesion is generally the result of a hematogenous infection, and although the infecting organism may not be responsible for the primary focus, it may nevertheless gain access to the blood stream from such focus. The finding of the same organism in both lesions is not absolute proof that the primary focus is the real cause, or the only cause, as there may be other foci through which the same organism may enter the circulation.

Pus formation, in and of itself, is confined almost exclusively to the liquefaction of inflammatory exudates, often releasing and

destroying considerable areas of forming granulation tissue. The epiphenomenon injected here is found to be saprophytic organisms which decompose the pus, forming its very irritant qualities.

The point in this particular consideration is this. We have suppurations occurring in the mouth that are simple in their form — that is to say, the micro-organisms producing pus are the only ones active in the disease. They are indeed the disease-producing power in these cases, and if they act alone they may produce no very great systemic disturbance. But in many cases an epiphenomenon is ingrafted upon this disease by the introduction of saprophytic micro-organisms which decompose the pus formed; or other organisms may enter the focus and these may become the most important element in the establishment of the secondary lesions.

This interference with the ordinary run of pus formation is especially common in the infections of the mouth, for the reason that the mouth secretions are constantly loaded with a large flora. These will grow for a time in the mouth secretions and may be included with those which cause the suppurations. These may be pathogenic varieties which will increase the difficulties by engrafting their effects upon those of ordinary pus-producing forms.

The saprophytic organisms never grow in living tissues, but only in the fluids which have been separated from living tissues. They decompose these fluids, and in the decomposition, products are formed which are poisonous and are absorbed into the general system. This has been called blood poisoning. Many examples of this occur in the fleshy foods eaten which have begun decomposition that is not noticed. The ingestion of such food will carry with it this poison and make the patient very sick for a time. In general theory its action is like a dose of poisonous medicine. It comes quickly and sharply, and if the patient survives its action, it gradually passes away — that is, when the poison is once expended, it is done. This is generally spoken of as ptomain poisoning. But such an organism growing in pus that is being produced continuously, will grow for a time, producing a systemic condition by a slow absorption of these products, which is often very serious. Therefore treatment may be for the prevention of these saprophytic growths, or their eradication when present, as well as for the cure of the pus formation itself.

THREE GROUPS OF CHRONIC FOCI IN THE MOUTH.

In the mouth there are three groups of chronic foci: (1) Deposits of salivary calculus, with which may be included certain fillings, crowns, bridges, etc., which impinge on the gingivæ and keep them in a state of constant low-grade inflammation; (2) pus pockets alongside the roots; (3) chronic alveolar abscess.

Of the three groups of foci, it is important to note that the conditions presenting in the first group are such that the focus may be easily eradicated by the treatment which has already been mentioned. The removal of the deposits permits the inflamed tissue to recover almost immediately. It is then a question of preventing new deposits. The removal or modification of fillings, crowns, etc., avoids future irritation and eliminates the focus, in cases in which pockets have not already been formed alongside the roots.

The pus pocket is best adapted for catching all of the numerous organisms which may be floating in the mouth, and transmitting them to the blood stream. The granulation tissue overlying the roots is very soft, and new blood vessels are being constantly formed and destroyed, presenting extraordinary opportunities for the hematogenous organisms within the pocket to enter the circulation.

The peridental membrane is detached from the cementum by suppuration, and this becomes the important factor in maintaining the chronicity. As a part of this process the cementoblasts which overlie the surface of the cementum within the peridental membrane are also destroyed. These are the only cells which could cause reattachment of that tissue. Soon after detachment, the fibers of the peridental membrane, which formerly passed from the cementum of the detached area to the bone, disappear, and a little later the bone to which they were attached is absorbed. Thus, in addition to the fact that the denuded cementum is dead, all of the specialized elements necessary to the connection of root with bone are lost, and a reattachment of this tissue to the cementum of the root can not take place. Therefore pockets remain about such teeth and are subject to frequent reinfection.

In the case of the chronic alveolar abscess, the chronicity may be maintained by the dead pulp, if it remains in the tooth, or by the denuded cementum about the end of the root. In cases in which the peridental membrane about the apex is not destroyed, the treatment of the root canal should eliminate the focus. If

the cementum has been denuded, the condition is practically the same as the pus pocket which is open at the gingival line, so far as the continued chronicity is concerned, as there is no possibility of a reattachment. The chronic abscess has not the same exposure to the fluids of the mouth as has the pus pocket, and does not present the same opportunity for direct reinfection.

We should not, therefore, expect vaccines to have more than temporary effect on the local focus, because there does not remain the physiologic possibility of repair. The dental treatment indicated is the elimination of the focus in the case of the alveolar abscess, which has destroyed the attachment of the membrane, by resecting the denuded root end or the extraction of the tooth. If it is a pocket alongside the root, exposed to the fluids of the mouth, the tooth must be extracted, or palliative treatment employed which will be effective against reinfections.

The pus and products of putrefaction and various organisms which may be present, especially the hematogenous varieties, are entering the blood through the thin-walled vessels in the loosened tissues, which are in a constant state of chronic inflammation of low degree. The lymphatic system is also taking up its quota of these poisonous materials, and the small lymphatic glands at the angles of the neck are often in a chronic state of mild, or more considerable, enlargement. Under these conditions the general health of the robust patient is put to a test of its strength to maintain itself without notable impairment. Many persons of the weaker sort become subjects of a low degree of septicemia, which spreads its peculiar pallor over their countenances, saddens them, and they lead lives constantly bordering upon actual illness. And not a few of these succumb to some intercurrent disease, which, but for the condition within the mouth, and its general systemic effect, they would easily have withstood.

Defense by the tissues.

It should be kept in mind that the natural resistance of the tissues is inclined to prevent or retard the occurrence of systemic lesions from chronic foci. Probably no tissue is more vigorous in resisting infection than the mucous membrane of the mouth. The presence of such a focus does not indicate that the individual is suffering from systemic effects, but he is undoubtedly in constant danger. Certainly many people, probably the majority of those who have such foci in the mouth, will continue in excellent health, or apparently so, for years. There may

never be an indication that their health and vigor have been impaired.

On the other hand, it should be remembered that the definite secondary lesions resulting from these foci are of such gradual development that they are generally not recognized by the patient, and do not come to the attention of the physician until they have made such progress as to be incurable, or at least very obstinate in their amenability to treatment. This fact demands the eradication of the foci for the protection of the health of all persons, whether apparently suffering or not.

THE DENTIST'S OPPORTUNITY.

The opportunity before the dental profession to take an important part in the preservation of the general health is almost without parallel in medical advancement. There seems to be no question but that the mouth contains more such foci than all other regions of the body combined. The secondary effects of these foci present great difficulties to the physician in treatment. The foci are easily recognizable by thorough mouth examinations, which must be made by the dentist. The means of protecting the general health in their treatment are simple. Success depends upon a full understanding of the situation by both physician and dentist, together with the education of our people to the danger from the primary focus, the insidious progress of the secondary effect, and its intractableness to treatment.

The dentist should apply treatment on the basis of a careful diagnosis and his knowledge of the danger to the person who apparently is in perfect health. This is preventive practice in the highest degree. Eradication of mouth foci should not be delayed until secondary effects have become manifest. Every dentist, who has a full appreciation of the situation, will realize that, in the management of cases, he is doing his highest duty to humanity in the preservation of health by keeping the mouths under his care free from these centers of distribution of infection.

SUMMARY.

In the light of our present knowledge, we are justified in making the following summary of the relation of mouth foci to general systemic conditions:

1. The mouth contains a large variety of micro-organisms, which may be divided into two groups; those which are normal

or constantly present, and those occasionally or frequently found.

2. Conditions in the mouth are such that slight inflammations of the gingivæ are of frequent occurrence, being present in about ninety-five per cent of mouths of adults.

3. These slight inflammations, if untreated, may gradually progress to chronic suppurations. The suppurations are caused by organisms normal to the mouth.

4. All organisms in the mouth, whether normal or accidentals, have access to the blood stream through the soft granulations.

5. The normal resistance tends to prevent systemic effects and is apparently successful in the large majority of cases.

6. The transmission of infection from the primary focus is principally hematogenous.

7. The primary focus is characterized by suppuration, while the secondary lesion is non-suppurative. Therefore the secondary lesion is not caused by the principal organism of the primary focus, but by other organisms which enter the primary focus with or after the pus producer, and thus gain access to the circulation; or else the morphology of the pyogenic organism is changed if it produces the secondary lesion.

8. The organisms entering the circulation through such foci appear to have an as yet unexplained tendency to locate in particular tissues.

9. The secondary effects include a very wide range of conditions. Chronic arthritis, endocarditis, nephritis, cholecystitis, ulcers of the stomach, and appendicitis are the most frequent definite lesions. General impairment of health and vigor, with or without recognizable lesions, is common.

10. The secondary effects are usually insidious in their onset and progress, and, when cases present to the physicians for treatment, are difficult of management.

11. It is imperative that the primary foci be eliminated, regardless of the apparent systemic effect or lack of systemic effect.

12. For the reason that the mouth contains the primary foci in the large majority of cases, a great opportunity is open to the dental profession to prevent grave systemic disease.

ORAL PROPHYLAXIS

PROPHYLAXIS may be defined as preventive treatment for disease, especially for a particular form of disease in an individual. Oral Prophylaxis should therefore include the treatment employed, mostly by the dentist, to prevent particular diseases in the mouths of individual patients. There should be recognized a danger of, or a tendency toward, a certain disease in the mouth of the individual, and the measures taken to prevent it constitute the practice of oral prophylaxis.

GENERAL PROPHYLAXIS.

The methods employed in the prevention of disease will differ with almost every disease with which we deal, and must be based upon an accurate knowledge of the causation of each. For instance, when the United States undertook the task of building the great Isthmian canal, the locality was found to be infested with yellow fever, and a much dreaded miasma, so called. The French had made an attempt to build the canal and failed, largely because so many of their men were rendered inefficient or died from these diseases. Our scientists had learned, while dealing with the Cuban proposition in the latter part of the nineties, that yellow fever was communicated to men by a certain species of mosquitoes (stegomyia) and also that the so-called miasma was communicated to men by another variety of the mosquito (anophiles).

The first thing our Government did in Panama was to place an army of men immune to yellow fever (by reason of having had the disease and recovered) into the zone to destroy the breeding places of these mosquitoes everywhere within a mosquito's flight of the zone of canal building. The prevention of the diseases has been practically complete.

In this way a veritable pesthole was quickly converted into a healthful place. Really very little sickness of any kind has occurred among the great army of men at work there. This is prophylaxis on a large scale. However, it is not a form of prophylaxis that would prevent the diseases that we are contending with in dentistry. Again, a serum has been discovered,

which quickly renders a child immune (temporarily) to diphtheria. Another immunizes against tetanus successfully, a dreadful form of disease from which few persons, if any, recovered before the use of the serum.

Personally, I have had no experiences with yellow fever, but with diphtheria I had a fairly wide experience before the discovery of the diphtheria serum, and it was sufficiently terrible to cause me to appreciate the value of the serum treatment. I also saw much of tetanus before the serum treatment came to our aid. It was certain death to the person attacked. One among the last cases with which I had to do makes a little story which I will relate. One morning a young physician came to me and asked me to go with him to see a patient, a boy about twelve years old, whom he had been called to see the day before. The boy had cut his great toe with an ax. The physician had stitched up the cut, which seemed unimportant. The next morning he happened to be passing the house and stepped in to see the boy. The little fellow seemed well enough, was going about the house, but was holding the sore toe up rather high from the floor. The doctor asked him to put it down on the floor. The boy said he couldn't. The doctor put it down with his hand, but it would not stay. He recognized at once that the case was unusual, and suspected tetanus, although he had never seen a case. He told the family that he would come back after a while, and came directly to see me, knowing that I had had experience with tetanus.

I saw the boy with him and found it undoubtedly a case of tetanus, just in the initial stage. All of such cases I had seen had ended in death after a struggle of the most desperate character. It was an ugly situation. Here was a boy attacked with a disease that would certainly cause his death unless something very unusual could be done. I had studied the disease carefully, had cultivated the micro-organism that caused it, and at the time felt that I knew all there was to be known about it, including the impotency of drugs to check it. The boy would die except one desperate chance might save him. The infection had not yet spread far. I proposed to the physician that he amputate the leg at the middle of the thigh within an hour. To this he assented. We called the parents and explained the whole matter as carefully as seemed possible. But say what we could, the father was simply furious. The idea that his boy, who seemed so well in every way and was at that moment playing cheerfully with some traps he had been constructing, watching the working

of the triggers, should be required to submit to such an operation, could not be entertained by him. Other counsel was sent for, and after careful examination, gave the same advice. It was no use. The parents would not have it. The next day the boy was not playing with his traps. The possibility of helping him by an amputation had passed. He died after eight days.

With diphtheria, which endangers whole neighborhoods of children, it is different. This disease is cut short in the child who is just becoming sick with it, and is prevented by immunizing other children who are as yet well. This is prophylaxis in a true sense.

At one time I was occupied in the management of epidemics of typhoid fever. In this it was different again, and it depended upon a much wider knowledge of the source of infection. In cases where the spread of the disease depended upon sick persons coming into a neighborhood and spreading it, all that was necessary was to stop the transmission from one to another, and to see that the minutiæ of the nursing was properly done. In this the greatest difficulty was to bring people to an understanding of the situation and secure their coöperation by compliance with regulations prescribed. The source of distribution of infection — in the ground, in the water, milk or other food — had to be found and corrected.

This is perhaps enough to give a full understanding of what prophylaxis, or the prevention of a specified disease, means. Prophylactic treatment, like all other treatment, should be based on a thorough knowledge of the etiology and pathology of the disease under consideration. For most diseases, the treatment employed has naturally become more effective as knowledge of the disease became more complete; treatment has at first been for the alleviation of pain or some distressing symptom, and later has gradually been directed toward the underlying cause or condition. When the complete pathology and etiology have been worked out, it has often been possible to apply preventive treatment, as is now done in diphtheria, tetanus, typhoid, etc. Preventive treatment can usually be employed when the etiology is fully understood.

ORAL PROPHYLAXIS.

Oral Prophylaxis includes the treatment employed to prevent particular diseases in the mouths of individual patients. In its broadest sense, this includes the prophylactic phase of practically every operation which the dentist performs. In a

more restricted use of the term, it has been commonly applied to a more or less systematic plan of thoroughly cleaning and polishing the surfaces of the teeth at stated intervals.

Such a large proportion of our people are subject to dental caries, alveolar abscess and diseases of the peridental membrane, and the consequences of chronic foci of infection in the mouth have been shown to be so far-reaching, that more definite prophylactic measures should be very generally studied and employed. These may well be divided, for each condition, into those which should be a part of our routine dental operations, and those technical procedures which may be employed independently. For example, in the placing of a proximo-occlusal filling in a molar tooth, the operator should be careful, in the preparation of the cavity, neither to expose the pulp nor to cut so closely to it that it will be in danger of death from thermal shock. This is prophylaxis against the death of the pulp and alveolar abscess. Also, in the preparation of the cavity, the rules of extension for prevention should be followed in obtaining the outline form, thus placing the margins of the filling in self-cleansing positions and thereby preventing a recurrence of decay. This is prophylaxis against dental caries. Further, in the placing of the filling, the proximal surface should be finished to proper form with a good contact, so that the interproximal gum septum will be protected from injury by food lodgments. Thus a recurrence of decay may be prevented in the tooth filled, a beginning of decay may be prevented in the proximal tooth, and an inflammation of the septal tissue may be avoided. This is prophylaxis against both caries and peridental disease.

In presenting the subject of oral prophylaxis, we will consider separately those procedures which may be employed against dental caries, and against diseases of the peridental membrane. It is recognized that the prevention of dental caries, and the prompt treatment of decays which have occurred, including the precautions for preventing inflammations of the pulp, which have been mentioned, constitute the highest degree of prophylaxis which may be employed against diseases of the dental pulp and their sequelæ — acute and chronic alveolar abscess, necrosis, etc. It is impracticable, in this writing, to mention all of the prophylactic phases of our routine dental operations in relation to these various conditions. Therefore, we will present here, for each condition, only those technical procedures which may be employed in what has been commonly termed prophylactic treatments. A grouping of these will show

that we are justified in following a more or less definite technic for prophylactic treatments, with certain modifications for each individual. Subsequently a systematic plan of mouth hygiene will be presented.

THE ORAL PROPHYLAXIS TREATMENT, SO CALLED.

For a number of years past there has been much talk of prophylaxis in dentistry. The principal credit for attracting the attention of the profession to this subject is due Dr. D. D. Smith, of Philadelphia. The plan suggested for preventing the occurrence of disease in the mouth, including caries and diseases of the gingivæ, is that the patient visit the dentist at stated times, varying from once every week or ten days to once every month or so, in order that every surface of every tooth may be scoured with pumice on the end of a stick of orangewood or similar substance. For proximal surfaces, strips of some delicate fabric are used.

This idea of prophylaxis had a considerable following for a number of years and is still practiced by some very good men. A considerable part of this service came to be turned over to young women assistants, who were especially trained for it. What was done by the patient in the cleaning processes in the interim between visits to the dentist, has never been so clearly shown, but it is safe to say that the patient was trained in the care of the mouth between visits to the dentist.

It has been my judgment that this plan of prophylaxis was much too heavy in its continuous operation to be successful. Also, that if it were kept up, as represented, much harm would be done to the margins of the gingivæ by the scrubbing with pumice on a stick, and probably greater harm still to the attachment of the soft tissue at the gingival line by the use of tapes so frequently as once per month for a number of years together. Recently we are hearing much less of this plan of prophylaxis in dentistry. I strongly suspect that one of the difficulties found has been to keep patients sufficiently responsive to their engagements for these cleaning processes. However, the plan has in it the phase of continuity which has a high value.

In the management of the diseases with which we are contending, the practical procedures in oral prophylaxis should take their place in the routine care of the mouth of each individual, having their proper relation to other operations, and both should be supplemented by proper training of patients in a systematic plan of mouth hygiene. No one of these elements in the care of the mouth can be set apart and be successfully employed alone in

many cases. For most people, there is such an interdependence of what may be termed the regular dental operations, oral prophylaxis and mouth hygiene, that all must be employed if the mouth is to be maintained in the best state of health..

The technic of the prophylactic treatment is simple. It consists of the very thorough scouring and cleaning of the surface of the enamel, and of denuded root surfaces. It should not as a rule go further than this. A limited number of very shallow penetrations of the enamel by caries, particularly in gingival third positions on buccal and labial surfaces, should be treated by grinding away some of this enamel to make the surface smooth. The very radical practice recommended by a few, of grinding away the outer surface of undecayed enamel, is to be condemned.

Application to dental caries. I have already called attention to the fact that in its broader meaning, we are, or should be, applying oral prophylaxis against dental caries in almost every operation which we perform. With each operation we will, to a greater or less measure, help to prevent the progress of caries in the particular mouth, if we are giving our patients the best possible service.

But we are to consider what may be accomplished by what we have designated as the oral prophylaxis treatment to prevent the inception, or progress, of dental caries. In the application of this treatment, as of any other, we must have the clearest possible understanding of the pathological problems involved.

So far as the treatment of caries is concerned, this service has been undertaken by those who have specialized in it, under the slogan that "clean teeth do not decay." This means that by such treatment the teeth must be kept clean, and the question naturally arises, how frequently must such a treatment be given in order to keep the teeth sufficiently clean. The answer must be that it will depend on the susceptibility or immunity of the individual. Some persons are so absolutely immune to caries that such treatment would never be necessary against caries, while others are so susceptible that I can hardly conceive of cleanings at intervals sufficiently close to be effective in actually preventing caries.

The general rule followed in this work has been to have most patients come in every two to four weeks for a "treatment." Is that often enough? If we are to depend on this alone, it certainly is not for most people. If it is supplemented by careful cleaning, two or three times a day, by the patient, it should be very beneficial, though it is a mistake to promise patients that

decay will be prevented. It may be prevented; it should be retarded in proportion to the frequency of the treatments. The principal difficulty encountered in this service is to keep the patients coming. It's like the old story of our Arkansas friend whose house needed shingling — he couldn't do it while it was raining, and it didn't really need it when the weather was clear. The dentist must have a strong hold upon his patients to keep them coming month after month, year in and year out. This is for many the most difficult phase of the whole plan. A few men have apparently been very successful with this plan. There is some question as to how long they may continue. Most dentists have not been successful with more than a very limited number of persons.

It should be understood, then, that we are to employ all other means at our command in treatment, and that we are not to endeavor to prevent dental caries by prophylactic treatments alone; that we are also to use our utmost endeavors to secure the best possible coöperation on the part of the patient in the procedures which will be mentioned under Mouth Hygiene. In other words, we will consider the possibilities of the oral prophylaxis treatment as a part of our daily service.

In the prevention of caries, we have only the beginnings in enamel and the surface spreading on enamel to deal with. We are not concerned with caries of dentin. We know from our studies of pathology that there are three groups into which we may place practically all beginnings of caries: (1) Decays occurring in defects in the enamel — pits and fissures in the occlusal surfaces of bicuspids and molars, buccal surfaces of molars and lingual surfaces of upper lateral incisors; (2) decays occurring in the proximal surfaces of the teeth; (3) decays occurring in the gingival third of buccal, labial and occasionally lingual surfaces. Even in the susceptible person all other areas are practically immune and require no treatment beyond the natural cleaning which they receive in mastication.

Let us then consider these three groups separately and see in what measure the prophylactic treatments may be effective.

Pit and fissure decays. These occur in defects in surfaces which are otherwise kept clean by mastication. The defects are such that micro-organisms may grow in them without being disturbed unless artificially removed. The acid formed in the deeper portions is directly applied to the enamel. The majority of these decays occur early, soon after the teeth erupt. It would seem reasonable to conclude that these decays would require the

most aggressive efforts, both as to the detail and frequency of the cleaning, to be successful. The dentist could hardly expect to prevent decay in such positions by thorough cleaning as often as once a week. It would require the earnest coöperation of the patient by proper daily care. And the effort must be continued from the earliest time in the childhood period of the permanent teeth to and, for most persons, beyond even middle age; for almost the lifetime.

Even supposing that we might be successful, is it worth the effort, when we can by a simple filling operation so change the conditions for each such area as to make it self-cleansing and remove practically for all time, providing our filling is well made, the danger of decay in the surface? Is it not a better procedure, from the prophylactic viewpoint, to place a filling on the first slight appearance of decay and end the matter? On the other hand, the effort should be made to prevent decays in these defects, as part of the treatments applied to other areas, up to the time when decay first appears, but they should then be promptly filled. However, they certainly do not present conditions which favor success by the method of prophylactic treatments alone, or even the combination of prophylactic treatments and mouth hygiene.

Proximal decays. Proximal decays occur on smooth surfaces of enamel just to the gingival of the contact point. The majority of these occur between the tenth and twenty-fifth year. Normally the crest of the septal gingivæ should reach practically to the contact point and thus protect the proximal surfaces from beginnings of caries. It is only when there has been a slight recession of this crest that the opportunity is given for the attachment of a colony of micro-organisms, and a beginning of decay. Therefore, in any procedure, either by an operation such as filling, or in prophylaxis, every care should be exercised not to injure the septal tissue, because injury usually results in recession.

There is little question but that sufficiently frequent thorough cleansing of these areas will prevent decay. The required frequency will vary with the susceptibility of the individual. The technic of cleaning must be one which will not injure the septal tissue and cause, by the necessarily frequent repetitions, a gradual recession of that tissue, which would increase the size of the area of liability on both proximating teeth.

A silk ligature which has been loaded with pumice, by first moistening an inch or so of it and then pressing it in the pumice, is oftentimes the best means. It has no sharp edge to cut the

gingivæ and may be drawn back and forth by being held first against the mesial surface of the one tooth, and then against the distal surface of the other. A very narrow linen tape may be used in the same way, although it is more apt to injure the gingivæ. Tapes with knots, etc., are made for this purpose, but most of them are too wide. So far as the treatment of caries is concerned, it should be remembered that colonies of micro-organisms do not grow on the enamel underneath the healthy gum margin and it is therefore only necessary to polish the *exposed* surface of the enamel; no polishing of subgingival spaces is indicated. In some interproximal spaces, a thin orangewood stick may be used to advantage; in most there is not sufficient room to permit of its use without injury to the septal tissue. Under Mouth Hygiene we will consider the question of the daily cleaning of these areas by the patient.

I should say that there seems to be more reason why oral prophylaxis treatments should be applied for this class of decays than any other, at the same time there is the greatest possibility of serious injury to the soft tissues.

Gingival third decays. These occur in fewer mouths and later in life than other decays, most of them after the twentieth year. They occur in positions which are so easily reached by the tooth-brush in the hands of the patient that it would seem unnecessary for the dentist to undertake to treat them by cleaning operations. It has been satisfactorily demonstrated that the patient can prevent these decays by proper brushing, and there is much room for question as to the effectiveness of cleaning by the dentist alone. In other words, unless the dentist has the coöperation of the patient by proper brushing, he will not succeed; and if these areas are properly brushed by the patient, his services are unnecessary.

Cases which present with beginning decays of very slight depth in these positions may be ground smooth and polished to facilitate the cleaning. Grinding should not be done unless there is an actual beginning of caries.

There is no question but that a monthly cleaning by the dentist will do much toward keeping the patient active in doing his part. The monthly cleaning by the dentist is probably of less real value than the monthly criticism of the patient's care as a stimulus to the patient to take better and continuous care of the teeth. This is to my mind the most important feature of the oral prophylaxis treatment for dental caries.

Application to diseases of the peridental membrane. As disease of the peridental membrane practically never occurs without a preceding gingivitis, as has been discussed fully in the consideration of the etiology and pathology of these diseases, our attention must be given, in the application or prophylactic treatments, to the inflammations of the gingivæ. The causes of the common inflammations of the gingivæ may be grouped under three headings: (1) Deposits of salivary calculus; (2) Deposits of serumal calculus; (3) Injuries.

Gingivitis caused by deposits of salivary calculus. A knowledge of the conditions under which deposits of salivary calculus occur is necessary to the employment of effective means of preventing inflammations from this cause. The deposit must be either prevented entirely, or, if it occurs, it must be removed before injury is caused by it. In the consideration of the subject, it has been shown that salivary calculus is deposited only (1) when calco-globulin is being secreted with the saliva, and (2) when the local opportunity — an irregular surface for lodgment — exists. The deposit seldom occurs in the mouths of children, but usually in adults, and increasing with greater age. It has been shown that the outpouring of the calco-globulin is in paroxysms a few hours after a meal and these may be repeated daily or even after each meal. The deposit occurs most frequently on the lingual surfaces of the lower incisors, where caries practically never occurs, and on the buccal surfaces of the upper molars in the gingival third. The deposit is very soft when first laid down — so soft that it may be easily brushed away; it gradually hardens so that within twenty-four hours it may be so hard that it can not be easily removed with a brush. It may be easily removed with the tooth-brush and plain water, if the brushing is thoroughly done twice a day. It would seem, therefore, that the patient ought to prevent these deposits; but as a matter of fact, most of them will fail in part, either by errors in the manipulation of the brush, or because of omissions in the twice a day routine. Therefore, a considerable percentage of our people should come more or less frequently — the interval to be adjusted to the individual — for the removal of these deposits and the polishing of the surfaces on which they have occurred. The effort should be to so manage these cases that there will be the least possible blunting of the crests of the gingivæ; for the broader the shelf which these offer, the greater will be the opportunity offered for the accumulation, and likewise the greater the difficulty in its removal with the brush.

Gingivitis caused by deposits of serumal calculus. These deposits occur on the enamel under the free margin of the gingivæ, or on the cementum in a pus pocket; never on an exposed surface. They almost never occur in the mouths of children, and usually in the mouths of persons beyond twenty years of age. As with salivary calculus, the deposits should either be prevented, or removed before serious inflammation is caused. Calcoglobulin is likely to be poured out into the subgingival spaces contemporaneously with its secretion with the saliva, in paroxysms a few hours after a meal. It is also soft when first deposited, but is not so located that it may be removed by brushing. However, there is no doubt but that the frequent washing of the subgingival spaces with a jet of water from the syringe will be of much benefit in retarding the accumulation. The principal advantage in both brushing and washing will be in keeping the gingivæ in the highest degree of health, thus reducing to the minimum the conditions conducive to the deposit.

Here again, however, most patients will fail in considerable part through errors in the use of the syringe or neglect, and their care should be supplemented by the service of the dentist. Regular intervals should be determined, after a study of the particular case, and accumulations should be removed. In the subsequent polishing of the areas, great care should be taken not to injure the gingivæ.

The same rules of treatment apply to cases in which there has been a detachment of the peridental membrane followed by a deposit on the cementum, to prevent, as far as may be, the recurrence of the deposit and the extension of the injury.

Gingivitis caused by injuries. For traumatic gingivitis — the oral prophylaxis treatment is not indicated, except possibly secondarily. In all such cases the prevention of the recurrence or continuation of the trauma should have first attention. If this can be done the gingivitis will usually disappear unless something has occurred secondarily which would keep up the inflammation. In all such cases the areas should be thoroughly scaled and polished, a record made, and the area watched and treated again, if necessary.

SUMMARY.

In all of this treatment of oral prophylaxis, I wish to very strongly emphasize the fact that there should always be a definite indication for the cleaning and polishing for each part or each area of each tooth which is so treated. It should be remembered

that most of the exposed portion of the tooth crown is so thoroughly cleansed in mastication that it requires no artificial cleaning. I have stated that the pit and fissure decays will usually be best cared for by fillings. There remain, then, for most people: the proximal surfaces in which decays occur mostly during the period from ten to twenty-five years of age; the gingival thirds of buccal and labial surfaces in which most decays occur after the twentieth year; the gingival thirds of the lingual surfaces of the lower incisors and buccal surfaces of molars on which deposits of salivary calculus occur, usually in the mouths of adults; and the subgingival enamel, on which deposits of serumal calculus occur, usually after the twentieth year.

The greatest care should be taken in all of this service to avoid injury to the gingivæ. I would consider it definitely wrong to polish the enamel of the subgingival spaces in the mouths of children as a part of a routine treatment, because there is no indication for so doing, and the frequent repetition of it is practically certain to injure the gingivæ. Likewise there is too much danger of injuring the septal tissue to justify the indiscriminate practice of polishing all proximal surfaces every few weeks. Discretion must be used in selecting cases.

In the mouths of few patients are we justified in following out the most thorough and complete prophylactic treatments. Some patients need areas polished to prevent caries, others for salivary calculus, still others for serumal calculus. Each generally involves different areas. It does not seem rational, therefore, that we should scour and polish every part of the enamel of every tooth, as some men have suggested, but we should apply treatment for each individual to the positions which our best judgment tells us it is indicated.

MOUTH HYGIENE

ILLUSTRATIONS: FIGURES 477-500.

HYGIENE is defined as that branch of medical science which relates to the preservation and improvement of health, both in individuals and in communities. It has no special relation to particular diseases, as is the case with prophylaxis.

Popular education.

Mouth Hygiene should include all measures employed, mostly by each individual for himself, under the direction of the dentist, to keep the mouth in the healthiest possible condition. Every one should know what rules should be followed and what results may be expected. There is presented a great problem in education; in spreading the knowledge that many serious diseases which result from mouth conditions may be prevented; that the more common diseases of the mouth, such as the decay of the teeth and the inflammations of the peridental membrane, may be largely controlled by simple methods of cleaning at regular intervals.

Let us look for a moment at the larger educational problem; later we will consider the technic of cleaning the mouth. Physicians, dentists and nurses are the teachers who must, in their daily contact with our people, educate them in such matters. However, material aid has come and will continue to come from other sources. The most noteworthy single effort to this end has found expression in the Forsyth Memorial in Boston. A fund of two million dollars has been devoted to the erection and endowment of a splendid building for the purpose of caring for the mouths of the school children of poor families in Boston, and for the larger purpose of educating the general public to the value of clean and healthy mouths. The Forsyth Memorial is not to be considered a charitable institution, but an educational one — an institution which will not only prove the value of the proper care of the mouth by demonstrations and clinical records, but will also be so directed and used as to eventually place the responsibility for the guardianship of the healthy body and the healthy mouth, especially of our children, upon the state and nation. The posi-

tion is taken that it is quite as much the duty of the state to look after the health of our children as it is to look after their education.

Attention has been called many times to the fact that our Government has expended enormous sums of money in studying those problems involved in the preservation of the health of our domestic animals, and in the raising of crops, because these are economic problems; but little effort has been made to study and prevent, or to teach our people how to prevent, the diseases which carry off untold thousands of our population every year. The commercial spirit of the age has led us, not only as a nation, but as individuals, to neglect health in order to get ahead financially. We should realize that there is greater economy in the growth and proper development of healthy individuals; that each child who is strong and well soon becomes one of the units in the development of those things toward which the human race is set; he is a producer; while the child who is weak and sickly becomes more and more one of the world's cares, a consumer who does not produce, and often one who may become a destroyer by entering the class of criminals and mental defectives.

We must look forward to the day when our Government will devote itself to the study of problems involved in the preservation of the health of our people; when we shall have a national Department of Health, which will consider human problems in much the same manner as the Department of Agriculture has studied and developed methods for the improvement of stock and crops.

Much information regarding mouth conditions has been gained during recent years from the examination of the mouths of our school children in many cities. Most of this work has been performed by dentists on their own initiative or under the direction of our various dental organizations. It is gradually becoming better systematized and its importance recognized by civic authorities. Several States employ dentists as regular staff members of the various eleemosynary institutions, and a large number of cities have recognized the value of dental service by making appropriations as a regular item in their annual budgets.

It is my object to present facts lying at the basis of practical mouth hygiene. Our people should have a better and broader knowledge of the conditions of the development of certain of the diseases of the mouth, and the means of preventing them. I have already said that within my personal observation the loss of teeth caused by deposits of salivary calculus has been diminished as

much as seventy-five per cent by wider knowledge of the necessary personal care. We ought soon to reduce this so that only a limited number of persons would be so injured. The plans of daily cleaning of teeth are found to be perfectly dependable in preventing injuries by salivary calculus, as well as diseases which depend upon unclean areas in the mouth for their inception and progress.

A good understanding of the cause of a disease is necessary to the adaptation of means for its prevention. Much is yet to be done in the instruction of dentists, as well as the laity, regarding the causes of diseases of the soft tissues of the mouth, before we can be sure of the virtue of proposed means for their prevention. Already the active teaching of the public as to what individuals should do in the cleaning of their teeth as a matter of intelligent care of their persons, seems to have begun in earnest.

In a paper on Constitutional Diseases Secondary to Local Infections,* Dr. C. H. Mayo has this striking sentence used near the close of his remarks on the prevention of disease: "The difference between the knowledge of the layman and the medical attendant, including the dentist, should not be too great. Medical progress may be stayed from time to time that the layman may be educated to certain truths of health; that he must first know, then desire, and then demand proper health conditions."

The work now being done in the instruction of our people regarding the maintenance of healthy mouth conditions is intended to bring them closer to the dentist in their knowledge of such things, and in this way assist in bringing about better coöperation between dentists and patients.

CARE OF THE MOUTH.

Temporary teeth. The care of the mouth should begin when the first of the deciduous teeth erupt. Previous to the eruption of the teeth, the mouth of the baby needs no care for cleanliness, as a rule. Some years ago, it was the practice of many physicians to direct that the mouth of the baby should be swabbed with a piece of gauze, wet with boric solution, after each nursing. There seems to be no indication for this, and I feel that the practice should be condemned. Care should be exercised, however, to have the breast or the rubber nipple clean before the baby nurses, as a prophylactic measure against a sore mouth.

The care of the deciduous teeth should be along the same

* Dental Review, Vol. 27, 1913, p. 281.

general lines as for the permanent teeth, except that during the period of the presence of the deciduous teeth, as well as during the childhood period of the permanent teeth, it is unusual to have deposits of either salivary or serumal calculus, and therefore, as the rule, no measures need be employed to prevent these accumulations. If, however, the examination should reveal deposits, care to eliminate these should at once be instituted. Likewise, less consideration need be given to the soft tissues during this same time, as they are not often seriously involved.

It is to prevent or retard the progress of dental caries during childhood that the mouth should receive attention. As soon as the first deciduous teeth have erupted, the use of the toothbrush should begin. The brush itself should be of the smallest size obtainable; soft enough not to injure the gums, yet stiff enough to be effective in cleaning. (See Figure 477.) The motions of the brush should be the same as for the adult, as will be described in detail later.

In the beginning, the child's mouth should be cared for by the mother or nurse, and the brushing should be done immediately after each meal. As the child grows older, it should be taught to do the brushing, under careful supervision, until it has acquired the necessary skill and has formed the habit of brushing the teeth as the first duty after each meal. Nothing is of greater importance to the future health of the mouth than for the child to form this habit of prompt and regular brushing of the teeth. The child that forms such a habit will come to appreciate the comfort of a habitually clean mouth and will not be likely to neglect the cleaning in after years.

The dentist should never overlook an opportunity to impress the importance of saving the deciduous teeth in order to avoid, as far as possible, the danger of disease and irregularities of the permanent teeth, which often interfere with the proper development of the face. It is probable that nothing is more effective in gaining and holding the interest of both parents and child in the care of the mouth than to direct their attention to dangers which beset the permanent teeth if the temporary teeth are neglected. In the past our people have not been alarmed at an extensive decay, or an exposed pulp, or an abscess of a deciduous tooth, because, they reason, this tooth will be lost in a year or so, anyhow. If, however, these conditions are recognized as a menace to the permanent teeth, and particularly if the symmetry of the face is endangered, the case takes on a more serious aspect, and

the active coöperation of all concerned is more likely to be obtained.

TECHNIC OF CLEANSING THE MOUTH.

In cleansing the mouth, one should have plenty of water — running water preferred — and in addition to a suitable tooth-brush there is often some advantage in the use of toothpicks, rubber bands, floss silk, a rubber bulb syringe, and, for those who wear artificial teeth, special plate brushes. The dentist should have each of the above to show to patients. The movements of the brush, the liability of missing some certain teeth, and the training necessary to the best use of the brush should often be illustrated by actual use.

I am especially partial to running water for cleaning the teeth. It may be arranged to be running from the faucet and out of the basin, carrying away all debris, and in this way keeping the water clean. Water that is a little warm is much pleasanter than either cold or very warm water. In the absence of running water an ordinary pan or wash-basin will serve the purpose very well. It is, however, quite a point to have plenty of water.

The mouth should first be thoroughly rinsed with water, by taking a considerable quantity into the mouth, closing the teeth and lips, and forcing the water back and forth through spaces between the teeth. When thoroughly done, this is one of the most effective means of cleansing that portion of these spaces which can not be reached with the brush.

THE TOOTH-BRUSH. The brush is of more importance than all else in the cleaning of the mouth. The brush should be carefully selected. Most tooth-brushes are too large to permit of sufficiently free movement in the mouth. Brushes shown in Figures 480, 481, 482, 488 and 497 are too large. A brush about the size of those sold as a youth's brush is to be preferred. (See Figures 479, 483, 484, 487, 490, 491, 493, 494 and 495.) The bristles should not be too closely set; there should be nearly as much space between the rows of bristles, as is occupied by the bristles. (See Figures 477 to 480.) A brush having the bristles rather deeply notched crosswise is very desirable. (See Figure 496.) As to the stiffness, each person should select a brush which is as stiff as may be used vigorously on the gums without causing pain. Such brushes are usually marked "medium." A new brush is always somewhat stiffer than it will be after it has been used for a time. In buying, one should rather consider

what the brush will be after a little use. While the brush is new the bristles may be softened by soaking the brush for a few minutes in warm water before using. Persons who use too stiff a brush are apt to avoid brushing the soft tissues, and lose that which is of great value in cleaning, i. e., the stimulation of the gingivæ.

For many persons, special brushes should be selected to meet the peculiar conditions presenting in their mouths. In some mouths it is almost impossible to clean about the third molars, either upper or lower, unless the bristles near the end of the brush are very short. In many mouths, when open, the ramus of the lower jaw is so close to the buccal surfaces of the upper molars as to interfere with the movements of the brush which are necessary to clean these surfaces, unless the bristles are very short. (See Figures 493 and 494.) Those brushes with a tuft of very long bristles near the end are generally to be avoided, as they fold back against the handle, when the brush is carried far back in the mouth, and do little or no cleaning. (See Figure 497.) The end bristles should be short enough so that there will be sufficient room between the teeth and the cheek for the brush to be moved freely. Some of the brushes on the market, which have the long tuft of bristles at the end, make very good brushes if these long bristles are cut short with a pair of scissors. (See Figures 481 and 482.) In some mouths there is insufficient room to the buccal of the upper third molars for a brush with the shortest possible bristles. In such cases, a brush with a small tuft of bristles at the end may be carried directly across the occlusal surface, with the tuft reaching over. The tuft may thus be moved along the buccal and also the distal surface. (See Figure 496.)

For some persons, whose lower front teeth are inclined lingually, a brush with a bend in the handle (the back side of the handle being convex) will make it possible to reach the lingual surfaces of these teeth to better advantage. (See Figures 491 and 492.)

The dentist should examine each mouth as to the available space for the brush in various parts, especially about the third molars and the lingual of the lower incisors. He should also note whether it is best to have the jaws apart or the teeth in occlusion. In many mouths the buccal surfaces of the upper second and third molars can be cleaned more effectively with the jaws closed. The dentist should give directions as to the style of brush to be used. In this, there should be certain stan-

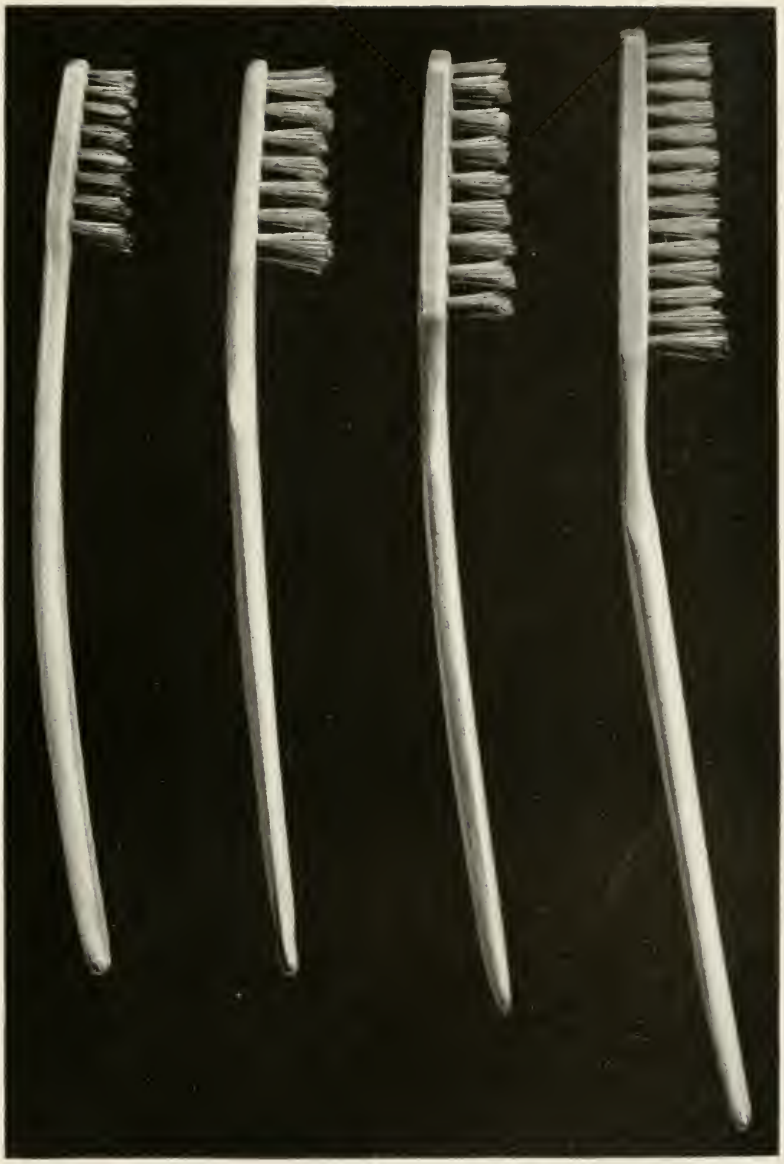

Fig. 477. Fig. 478. Fig. 479. Fig. 480.

Figs. 477, 478, 479, 480. Four tooth-brushes, actual sizes, for baby, child, youth and adult. All four have the bristles set sufficiently far apart and the brushes are generally of good form. The brush in Figure 479, ordinarily sold as a youth's size, is by far the best size for most adults, as there is better opportunity to manipulate it in the mouth. While some persons may use a brush as large as Figure 480, it is too large to permit of proper movements in most mouths.

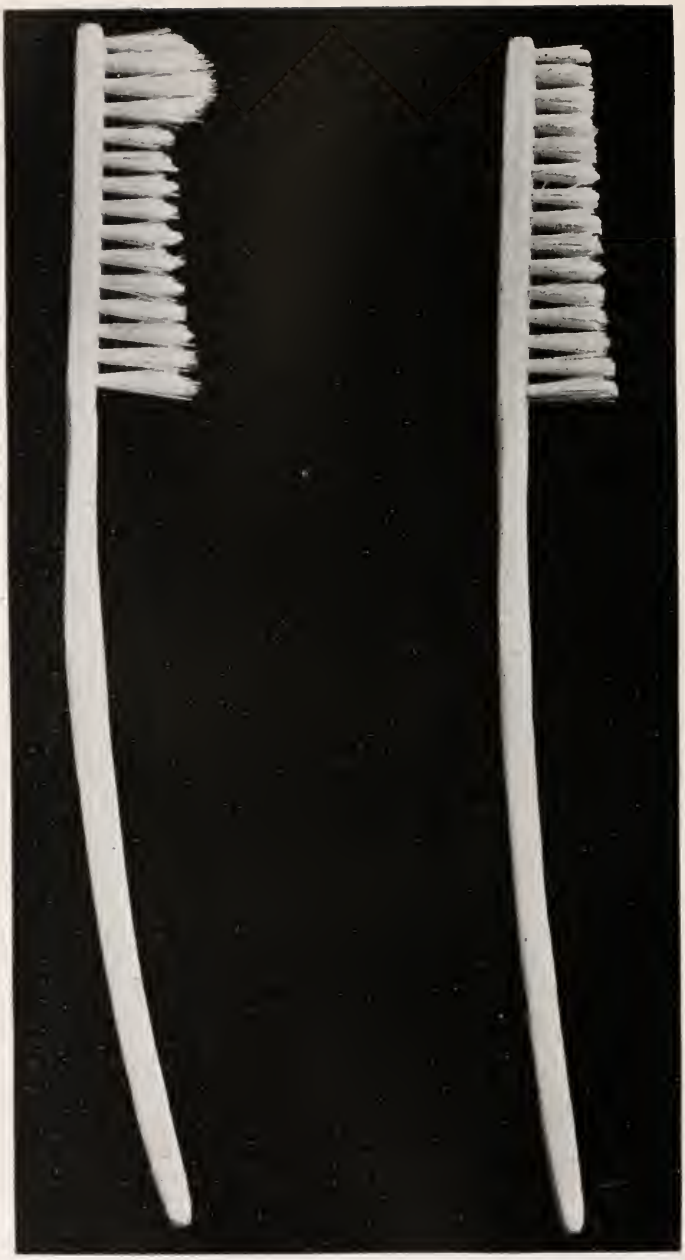

FIG. 481. FIG. 482.

FIGS. 481, 482. Two tooth-brushes, actual size. The brush shown in Figure 481 has the bristles well spaced. The tuft of longer bristles at the end reduces its effectiveness. (See Figure 497.) If these end bristles are cut off, as shown in Figure 482, it makes a much better brush. Both of these brushes are too large for most mouths.

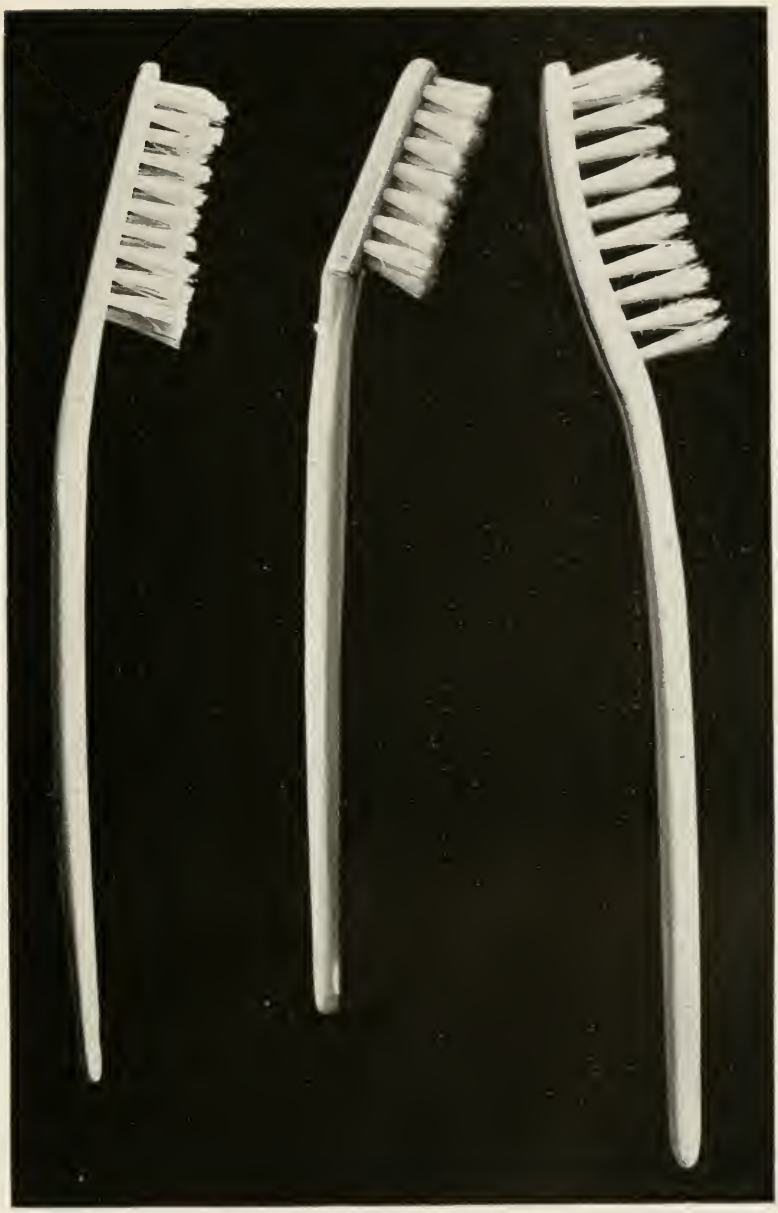

Fig. 483. Fig. 484. Fig. 485.

Figs. 483, 484, 485. Three tooth-brushes, actual size. Figure 483 is a splendid brush. This is the best form for most persons. Figure 484 is a similar brush with a greater angle, and is especially good for reaching the lingual surfaces of the lower incisors. (See Figure 492.) The spacing of the bristles in Figure 485 is very good, but the bristles are a little too long.

FIG. 486.

FIG. 486 shows the brush (handle cut off) in the proper position on the gum for the movement over the gingivæ and buccal surfaces of the lower teeth. The movement should always be from the gums over the gingivæ and teeth; this tends to keep the crests of the gingivæ thin, while the opposite movement tends to blunt the crests and thus give opportunity for lodgments.

dard types which will be right for most people, and others to suit special requirements.

Movements of the brush. Each person should form the habit of following a definite routine of brushing. It makes no particular difference where one begins or ends, if the habit is formed of following the same routine at each brushing. When one has once formed such a habit, there will be little danger of missing any part of the mouth. The following plan is suggested:

After the brush has been held in the water for a moment, the end should be carried fully back to the last molar tooth, or beyond it when possible, on the left side of the lower jaw. The ends of the bristles of the brush should be placed against the gums over the roots of the teeth, and then, with either a straight or a twisting motion of the wrist, the brush should be swept over the teeth toward their occlusal surfaces. (See Figure 486.) This should be done several times. One should learn to repeat this motion quickly with a twisting of the wrist, and while so doing, gradually bring the brush forward to the incisor region. Then, with fresh water, the brush should be carried back in the same way on the lower right side and the motions repeated, until all of the buccal and labial gum, gingivæ and the buccal surfaces of the teeth have been cleaned. These motions should be repeated for the upper jaw, the brush being first placed far back on the gum of the left side, and swept over the gingivæ and teeth in the direction of their occlusal surfaces. (See Figures 493, 494 and 495.)

Particular attention is called to the fact that the motion of the brush is first upon the gums, next over the gingivæ, next over the buccal surfaces of the teeth to the occlusal margins and off. Then the brush should be lifted and replaced upon the gums as before, and again swept over the gingivæ and the teeth, continuing until their occlusal surfaces are passed. One may soon learn to make these motions very rapidly.

As previously mentioned it will be difficult in many mouths to reach the gum overlying the upper third molars, or even the buccal surfaces of the crowns of these teeth, on account of the little space between them and the ramus of the lower jaw and the cheek, and a special brush will be necessary. (See Figures 493, 494 and 496.)

In those mouths in which the buccal surfaces of the back teeth can be more conveniently reached with the jaws closed, the brush should be used with a downward sweep from the upper gum over the buccal surfaces of the upper teeth, and a return

upward sweep from the lower gum over the buccal surfaces of the lower teeth.

Some persons seem not to effectively clean the gingival thirds of the buccal surfaces of the teeth with the upward and downward movements just mentioned. This will be more likely to be so, if for any reason the buccal gingivæ have lost their knife-like edge and are rather thick and blunt. Such areas should be cleaned with a forward and backward motion of the brush, about half of the width of the brush being over the gingivæ, the other half over the buccal surfaces of the teeth. I have occasionally seen some injury to the septal tissue by the use of this movement with too stiff a brush.

To go over the surfaces in each region three or four times, with the up and down motions, gradually bringing the brush forward to the front of the mouth, is sufficient to do all that can be done with the brush. These motions will bring the bristles in contact with every part of the tooth crown and through the embrasures as deeply as they can be readily forced.

The motions for the lingual surfaces of the lower molars should be practically the same as those for the buccal surfaces, but they are more difficult to make correctly. (See Figures 489 and 490.) The same may be said of the lingual surfaces of the upper molars. For both lowers and uppers, the brush must be held somewhat obliquely to the line of the arch and most of the brushing done with the bristles toward the end of the brush.

The brushing of the lingual surfaces of the incisors, both lower and upper, is best done with an endwise motion of the brush. (See Figures 491 and 492.) The handle of the brush should project out of the mouth parallel to the length of these teeth. The brush, while held in this position, should be placed on the gum and the motion of the brush should be over the gum, gingivæ and teeth. The brush should then be lifted, replaced on the gum, and the motion over gum, gingivæ and teeth repeated several times. This will tend to keep the crests of the gingivæ thin, while brushing with the opposite motion would tend to reduce the height of the gingivæ and thicken the crests. The brush may be moved from side to side across the lingual surfaces of the teeth close to the gum, for the removal of deposits which may have been missed by the other movement. This brushing should include the gum. It is well to call the attention of patients to the fact that they should feel the brush on the gum.

If there are defects in occlusal surfaces, these should also be thoroughly brushed. As a rule these surfaces are so well cleaned

in mastication that they require little brushing. If there are no defects, or if the pits and grooves have been made smooth by filling, they should need no brushing.

Teeth which are not used normally in mastication, either on account of the loss of opposing teeth, or on account of a sensitive or tender tooth, should be very thoroughly brushed on all surfaces, as the cleaning of mastication must always be regarded as the principal factor in mouth cleanliness.

Care of the brush. After using, the brush should be thoroughly rinsed with water and hung up to dry. If the brush selected does not have a hole in the handle, a brass screw-eye of proper size makes a very good holder. The eye should be large enough so that the handle can be slipped through it, the bristles being up.

After brushing, the mouth should be quite thoroughly rinsed with several mouthfuls of water. It is well also, to rinse the mouth a number of times while doing the brushing. The more effective rinsing, when the water is taken into the mouth, is done by closing the teeth firmly and forcing the water back and forth from lingual to buccal a number of times.

Until one has certainly formed the habit of brushing the teeth systematically in accordance with the plan mentioned, or any similar one which includes all positions which need to be brushed, he should stand before a mirror and watch every movement to see that the brushing is thoroughly done. Even those persons who take the utmost care in the brushing of their teeth are liable to miss one or more places, and the dentist should, in his examinations, be constantly on the lookout for such neglected places and call the attention of patients to them.

The toothpick. The toothpick is designed to cleanse the interproximal space and the proximal surfaces of the teeth. Normally the septal gingiva should fill the septal space completely to the contact point, so that there will be no opportunity for food to lodge. Decay of proximal surfaces usually begins only when the septal gingiva has receded a little. When it has receded, some effective means must be employed for cleaning the surfaces of the teeth between the contact point and the crest of the gingiva. This cleaning may be done with a toothpick, by passing the thin flat end of the pick between the teeth and rubbing the surface, first of one tooth, then the other. A toothpick made of quill, or as thin a wooden pick as may be had, and made of a very close-fibered wood, having a smooth hard surface without slivers or splinters, will be the best to use. If a quill pick is

used, the point should be cut off and the end rounded, to avoid pricking the gingivæ.

In connection with the use of the toothpick, one thing should be particularly remembered; that decays of proximal surfaces occur only when the gingivæ have receded, and that the repeated forcing of a toothpick between the teeth is very liable to cause the gingivæ to recede more and therefore may, in the long run, do more harm than good. Only those whose gums have already receded sufficiently to give room for a toothpick to be passed between the teeth without pressure on the gum, can use toothpicks without danger of causing further recession. (See Figures 209 and 210.) Few persons have this much recession before middle age; it is for this reason that younger persons should be very careful in the use of the toothpick.

Formerly I had been much opposed to the use of the wooden toothpick, because of more or less slivering of the wood in cutting them. In more recent years, however, many manufacturers have so improved the product as to eliminate the danger of injury to the gums from the slivers. One should, however, choose wood toothpicks very carefully, and buy only such as are smooth — actually polished all over — and free from slivers. There are still plenty of the cheaper sort to be avoided. It seems that every means yet devised for cleaning the proximal surfaces of the teeth must be used with care, lest the gingivæ be injured. A toothpick habit is liable to be formed in which the person will carry a wood toothpick sticking between certain teeth. This can not be continued long without seriously injuring the septal tissue.

Rubber bands and silk floss. Rubber bands or waxed floss silk are generally preferable to toothpicks for cleansing the spaces between the teeth. Neither should be used, except in those spaces where there has been slight recession of the gingivæ, or in spaces in which food may be caught occasionally, even though the gingivæ are normal. Certain foods, such as chicken and other stringy meats, are much more likely than others to slip past good contacts, or to slip into the septal space from the buccal, labial or lingual. A silk floss or a rubber band is usually necessary to remove such lodgments. It would not be a good plan for every person to pass a rubber band or floss silk between the teeth after each meal, because of the danger of irritating the gingivæ and causing them to recede.

In using either the rubber band or floss silk, it should be held with the fingers close up to the teeth on either side and then

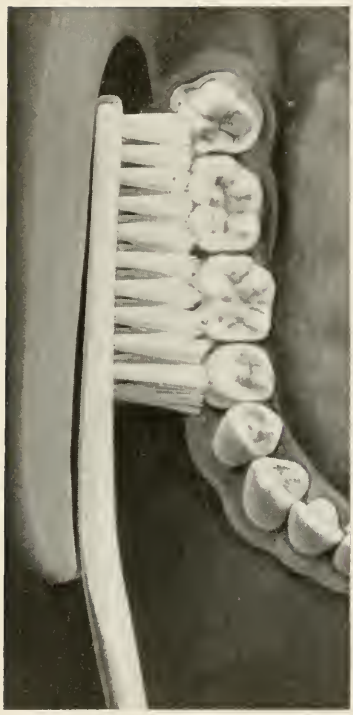

FIG. 487.

FIG. 488.

FIGS. 487, 488. "Youth's" size and "adult's" size brushes in position on the lower molars. It will be observed that the smaller brush is better suited, as it may be more easily manipulated in the mouth. The illustrations are actual size.

FIG. 489.

FIG. 490.

FIGS. 489, 490. These two illustrations show the position of the brush for the lingual surfaces of the lower bicuspids and molars. While the brush must be held diagonally to the line of the arch, the movement should be the same as on the buccal side. The brush should be placed on the gum and should then be swept occlusally over the gingivæ and teeth.

Fig. 491.

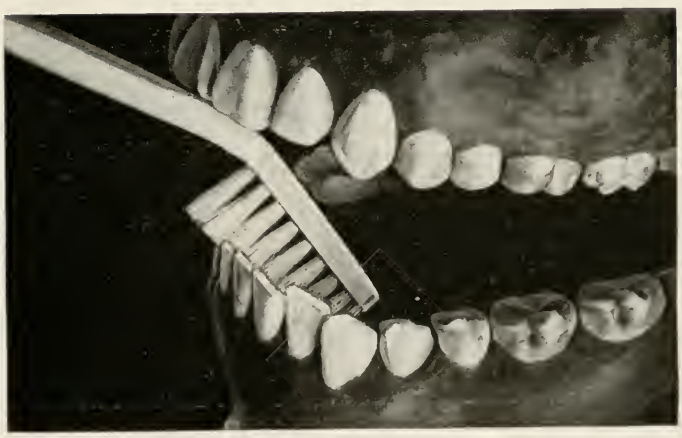

Fig. 492.

Figs. 491, 492. In brushing to the lingual of the lower incisors, the brush should be held parallel to the long axis of these teeth. Many people fail to brush the gums and gingival portions of the teeth in this position. If the brush has a handle bent as in Figure 492, the end bristles will touch the gum without the mouth being opened as wide as would be required with the brush in Figure 491. The bent handle is especially necessary if the lower incisors are inclined lingually.

Fig. 493.

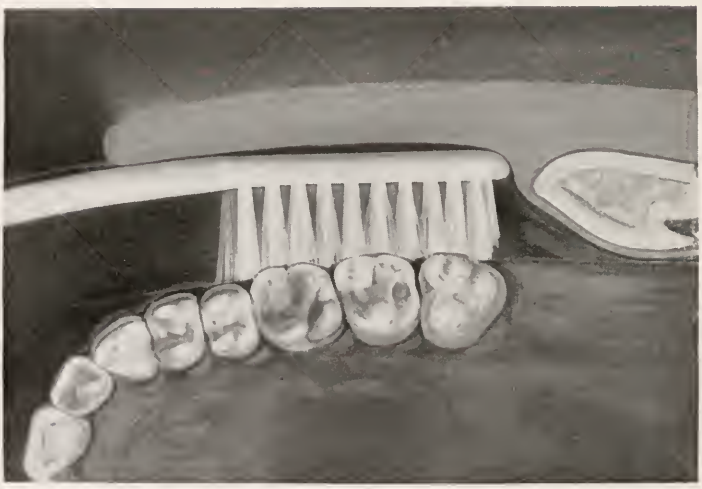

Fig. 494.

Figs. 493, 494. A "youth's" size brush to the buccal of the upper molars. In Figure 493 the ramus in section is shown in its position in many mouths when the mouth is open, preventing the brush from reaching the third molar. In Figure 494 the ramus is shown in its position when the mouth is closed, giving additional room for the brush.

carried very slowly past the contact point. In doing this the ends should be drawn a little, either to the mesial or distal, so that the silk or the band will pass to one side or the other of the crest of the septal gingiva as it passes the contact, thus avoiding injury to this tissue. After it is in the space between the teeth, the two ends should be drawn back and forth to rub the surface of one tooth, then it should be lifted over the crest of the gingiva and the surface of the other tooth rubbed in a similar way. It is quite a task to do this carefully and thoroughly in each of the thirty spaces between the teeth and few of those people whose teeth need such cleaning will do so regularly. It is, however, about the only effective method of cleaning these surfaces. Patients should be very carefully instructed in the manner of using the floss or rubber band, and should be impressed with the danger of injury to the soft tissues.

All interproximal spaces which require the use of a toothpick, rubber band or silk floss to remove food debris, should be carefully examined to determine the condition of the contact. In the majority of cases, unless the attachment of the peridental membrane has been seriously injured, it should be possible to correct either the form of the contact or the tightness of it, so that food will not be caught. The methods of so doing have been given elsewhere.

The syringe. It is often hard for me to say, when considering a case in practice, which is the more important for the patient to use, the brush or the water syringe. But there is no such thing as dispensing with the brush. The syringe should follow the use of the brush and the toothpick, or the rubber band, or the silk floss, whichever of the latter three may be employed. The syringe should be the last used. There are three principal uses for the syringe, (1) to maintain the health of the gingivæ by cleansing all of the subgingival spaces — the proximal subgingival spaces as well as those on the buccal, labial and lingual; (2) to remove food debris from interproximal spaces; (3) to cleanse pockets which may have occurred alongside the roots, as a result of detachment of the peridental membrane from the cementum. Food debris is not, as a rule, forced into the subgingival spaces, except occasionally on proximal surfaces. The object of washing out all of the subgingival spaces, while the gingivæ are in normal or nearly normal condition, is to thoroughly cleanse them by removing whatever residue may remain from the serum, often containing calco-globulin, which is constantly discharged into these spaces from that portion of the

gingivæ which overlies the enamel. There appears to be little residue in these spaces during childhood and early adult life, but as age advances, the content of this discharge is increasingly injurious, and the indication for washing the spaces is correspondingly increased.

For removing food debris from the interproximal spaces, the syringe is very efficient, and is especially indicated in those mouths in which there has been a recession of the septal gingivæ, while the contacts remain tight. In such cases the lodgments enter the spaces from the buccal or lingual and are not packed in, as is often the case when there is an open contact. This condition presents most frequently in the mouths of persons past middle age. The syringe is also of service in removing debris which has been forced past weak contacts, or crowded in where contacts are open.

Whenever detachments of the peridental membrane have occurred, the syringe offers the only means as yet suggested by which patients may clean these pockets and thereby keep the overlying soft tissue in the best possible condition. By frequent irrigation the inflammation may be controlled and the resultant outpouring of serum reduced to the minimum. The stream of water should be thrown with full power of the hand in compressing the bulb. There is no danger of using such force as will do injury.

The syringe should have as large a rubber bulb as can be conveniently held in the hand. The nozzle should be long enough so that the end will easily reach the third molars, and the opening in the end should be large enough to give a good strong stream. The bulb of the syringe shown in Figure 498 holds one and one-half ounces, the nozzle is three inches long, and the diameter of the opening in the end is 1.5 mm. Figure 187 shows one of these syringes, actual size. Aside from the need of having a large opening, it is important that the end of the nozzle be so large that it would be impossible to get it under the free margin of the gingivæ. In addition to its effectiveness in cleaning, a very important advantage of the syringe is that there is no danger of injury to the gingivæ in its use. There is no other method of doing this cleaning which does not present considerable danger from frequent slight irritations. These have been pointed out in the consideration of the toothpick, silk floss and rubber band.

In using the syringe one should follow the same routine as with the brush, beginning, we will say, with the buccal surface

of the lower left third molar. The bulb should be pressed with full force, and the end of the nozzle should be drawn over the buccal surface of the tooth, halting slightly in the forward movement as the nozzle comes between the third and second molars, then over the second molar, halting again between the second and first molars, and continue this to the median line. With a little practice, one may gauge the movement so that this will use one syringeful of water. With a second syringeful, the nozzle should be placed on the buccal surface of the lower right third molar and drawn forward in a similar way. The buccal and labial surfaces of the upper teeth should be gone over in the same way; then the lingual surfaces of both the lower and upper teeth. This requires eight syringefuls of water. If one has two syringes, only about one-half the time will be necessary, for there need be no waiting for the bulb to fill with water. One will fill while the other is being used.

The nozzle of the syringe should continuously touch the teeth, and be so inclined as to force the water toward the gingivæ. (See Figure 498.) One edge of the nozzle should be close to or barely in contact with the margin of the gingivæ, but in no case should the effort be made to place the end of the nozzle under the free margin of the gingivæ, on account of the danger of injury to this tissue. The tongue and lips may be of assistance in guiding the end of the nozzle to the right positions. On the lingual side of the arch especially, the tip of the tongue will, after some practice, guide the end of the nozzle as it is passed around the arch, and thus facilitate the movement.

In using the syringe in this way, all parts of the teeth and all of the subgingival spaces will be washed. The water will generally find its way to the deepest parts of the septal subgingival spaces as well. Most patients will soon come to feel the *lifting* of the gingivæ as the water enters the subgingival spaces. This use of the syringe gives a sense of cleanness and comfort not obtainable in any other way. It is well worth the effort expended for this purpose alone, and persons who once learn this use of the syringe will gladly continue with it indefinitely. But in addition to this, it is really the best aid to the brush as a cleaning agent which we have.

The syringe should be used by all adults and young adults as a part of the regular routine cleaning of their mouths. This may be done with perfect freedom without injuring the soft tissues. As a rule young children are free from inflammations of the gingivæ, other than those caused by proximal decays

which permit impactions of food, or other causes which may be definitely and promptly remedied. It is not therefore necessary that young children use the syringe. The use of the syringe should be begun as soon as there is apparent the danger of beginning proximal decays.

It should be recognized that certain impactions of food will be held so tightly between the teeth that they can not be removed by the syringe and water alone. The dentist should be able to so modify the forms of proximal surfaces that practically all such impactions will be avoided.

When gingivæ are inflamed. If the gingivæ are sore and inflamed, as they will be after the removal of deposits of calculus, physiological salt solution should be used in the syringe instead of plain water, until all soreness is passed. Indeed, whenever there is an inflammation or a break in the tissues, the salt solution should be used, and the washing should be done in the most thorough manner. Such places in the mouth heal particularly well under this treatment. No antiseptics of any kind should be used.

In cases in which there are several or many pockets, physiological salt solution should be regularly used. Patients should be given detailed instructions as to the most practical and convenient methods for carrying out this work. Probably the most satisfactory plan will be for the patient to have a bottle of salt tablets and a glass which will hold eight ounces. The glass may be filled with warm water, and two salt tablets added, which will make the proper solution. (See Figure 177.)

It is necessary to suggest some such plan, for we can not expect many persons to do this washing twice daily, month after month, and year after year, unless it may be done with little inconvenience and without requiring too much time. As has been mentioned, the time may be materially reduced by having two syringes, one of which will be filling while the other is being used.

Mouth-washes, pastes and powders.

No mention has been made of mouth-washes, tooth-pastes, tooth-powders, etc. It is believed that these are of very little or no value; that everything can be accomplished with plain water and a brush and nothing is gained by the use of medicine. If there is no disease of the tissues of the mouth, certainly no medicine is indicated. If the mouth is thoroughly cleaned the sense of comfort will not be bettered by the pleasant taste of a mouth-

wash. There is no merit in the use of an antiseptic mouth-wash, as its effect is of but a few minutes duration. With the development of more complete knowledge of the etiology of dental caries, we have come to realize that the acidity of the saliva has nothing to do with decay and alkaline mouth-washes are not indicated to prevent decay. No solution taken into the mouth will have more than a very temporary effect and will be of no practical value.

Some children, and possibly a limited number of adults, may be induced to take better care of their mouths if a pleasant-tasting mouth-wash, or powder, or paste is prescribed. The idea has become so fixed in the minds of our people, as a result of advertising propaganda by manufacturers and the belief of many dentists that these preparations are beneficial, that it is not to be expected that the public, or the members of the profession, will quickly change their attitude, but in the light of our present knowledge, there should be a gradual change to a more rational view. In the meantime, members of the profession should thoroughly familiarize themselves with these problems, and exercise judgment in presenting the facts to their patients. The most that can be said for the majority of such preparations is that they are probably harmless, although there can be little doubt but that some are injurious. If it is believed that a patient, and particularly a child, will take better care of his mouth if a pleasant-tasting, harmless preparation is used, I would offer no objection.

Dentist should put mouth in condition.

It should be the first duty of the dentist to put the mouth in condition. If there are deposits of either salivary or serumal calculus, they should be removed; places where food lodges on account of decays, open contacts, imperfect previous operations, etc., should be corrected, and sensitive teeth should be cared for. Then the training of the patient in the care of the mouth should begin. The mouth should be put in the best possible condition for the vigorous use of the teeth and should be as free from inflammations as possible, so that thorough brushing will not cause pain, before systematic cleaning by the patient may be undertaken with hope of success.

I have frequently seen persons who have tried to use the brush over inflamed, sore and bleeding gingivæ, only to make an utter failure, because the tissues were too sensitive to be brushed. They did not appreciate the conditions which rendered the brush

ineffective. Often several days are required after scaling operations for the inflammation of the gingivæ to subside, and patients should be instructed not to use the brush during this time. As has been mentioned, the dentist should have such cases under his observation and care until the inflammation has subsided.

When cleaning should be done.

The teeth should be cleaned after each meal. To this rule there should be no exceptions. It is the only safe rule for the large majority of persons to follow. When followed in this way and carefully done, it insures a continuously healthy mouth, so far as deposits of salivary calculus and food debris are concerned. There is a special virtue in cleaning immediately after meals. To one who has formed that habit, the condition of the mouth annoys until the cleaning is done; it can not be forgotten.

Some people may neglect the care of their mouths for years before injury will result, but it will come, sooner or later, to a large percentage of such persons. It should be remembered that calculus is always deposited in a very soft form, and at once begins slowly to harden. At first it is easily brushed away, but if neglected for a day or two, it becomes too hard for this mode of removal. Cleaning at stated times is the one effective means of preserving a healthy mouth. It is within the reach of every person who becomes sufficiently interested to learn how, and will do it faithfully. Neglect for two days may bring conditions which will make the cleaning ineffective in certain positions, because of the hardening of lodgments.

Because of the effectiveness of regular and thorough cleaning, one must not conclude that the services of the dentist can be dispensed with. Nearly every person will fail in some part of the prescribed routine. They may, in the slow changes of their habits in cleaning the mouth, skip some essential place. The dentist should impress the importance of being regularly consulted, in order that such errors may be corrected. He should be ever watchful for opportunities to criticize, to the end that as many patients as possible may be gradually trained to clean their mouths with absolute thoroughness. Areas which are apparently not well cleaned should be pointed out and the habit of cleaning corrected. This service on the part of the dentist should never be regarded as trivial because it is in the form of advice; on the other hand, it should be reckoned as one of the most helpful services he can render.

Training in cleaning of the mouth. Every person, in forming habits of cleaning the mouth, should be under the direct supervision of the dentist. It is very necessary that the idea of habit be recognized in anything that is to be repeated over and over again in the same way. Correct habits in every particular are essential in this cleaning. In the formation of the habit, every part of the mouth which needs it should be properly cleaned.

No other person than the dentist should be regarded as fully competent to do this training, because no other person has the knowledge and skill in examination which renders him fit to determine whether or not the cleaning is in every way efficient. For this reason, patients who are not doing effective cleaning should be requested to bring their brushes to the dentist's office, in order that their use of them may be criticized.

As an example, I will report a case of a young lady who was anxious to clean her mouth properly. She had learned to use the brush with the correct motions, but in examining her mouth, I discovered that the labial surfaces of the left cuspids, both upper and lower, were not well cleaned. Each had a growth of micro-organisms about the crest of the gingivæ. If this had been on the right side, I should have known the cause at once. After a moment's thought, I asked the young lady if she was left-handed. She looked at me curiously, and said, "Why do you ask such a question?" I gave her a hand mirror and pointed out the areas which had not been brushed, and said, "You must have used your left hand and did not quite make ends meet." That is, beginning with the molar teeth on the right side of the mouth, she would carry the brushing around to the left lateral incisors; and then beginning on the left side she would brush the molars and bicuspids; the left cuspids were not brushed. This is an example of the errors which occur in the habits of cleaning the teeth. With the person who holds the brush with the right hand this failure would be on the right side of the mouth instead of the left. This patient acknowledged that she was left-handed, and declared that she would make "ends meet" in the future. It is only by this careful plan of examination and direction that we can bring some people, who are conscientious in their efforts, to be really thorough.

I will relate another case which I think will be of interest, particularly because it was the first serious case of disease of the peridental membrane which I treated absolutely without antiseptics.

A young lady came to see what I could do for her mouth, having been recommended to me by one of my very good patients. I found her mouth much inflamed from deposits of salivary calculus. I told her that I would not undertake the treatment of her case unless she was willing to follow my advice in every detail as to the future care of her mouth. To this she assented, after questioning me as to what I would require. She promised full consent and obedience, except to the extraction of a left lower third molar that was then very loose, on account of detachments of the peridental membrane. I told her that I would risk getting that out of the way. Her case was apparently one of long standing, though she claimed it was not, and I undertook the treatment with some misgivings as to the result. She proved to be an excellent patient.

After the removal of the calculus, I found the injury about individual teeth very unequal. In some, a part of the bony alveolar process was gone. In others the full bony process was standing. I determined to cut away the tissues from all of the teeth to nearly the depth of the worst injuries, of which there were a number much alike, in order that a new line of attachment of the gingivæ might be formed as nearly regular as possible, to facilitate cleaning. I thought that the crest of the new gingivæ might finally be about the height of the gingival line, grading to a somewhat lower level for a part.

After this operation, the tissues healed well. All pus formation was carried away at a dash, except about the very loose third molar, which she would not permit me to extract. To undertake the treatment of it by uncovering the suppurating areas would almost accomplish its removal, and especially on the buccal side, would involve the removal of more tissue, much of it hard bone, than I felt justified in doing for a tooth that would apparently fall away in a few months. The new line of gingivæ seemed well established, and was very satisfactory. After I had had the case under observation for several months, this patient was ordered to Europe to do certain work to which she was assigned, and did not return until the end of fifteen months. She then came to see me. I asked her when she had the loose tooth out, and she replied: "Don't ask me any questions until you have examined my mouth."

The next day I found the third molar standing in the row hard and fast, with less than one-third of the root in the tissues. She evidently could use this tooth in mastication much the same as the others, as she claimed. The tissue that I had

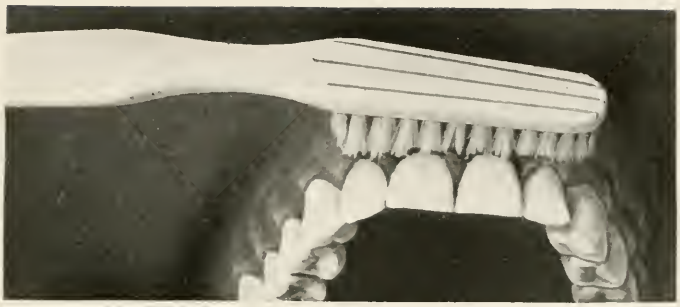

FIG. 495.

FIG. 496.

FIG. 495. The brush is shown in the proper position on the gum for the motion downward in brushing the upper front teeth.

FIG. 496. A little brush with a tuft of bristles on the end is desirable in many mouths to clean the buccal and distal surfaces of third molars by reaching over the occlusal surface. This is a brush designed by Dr. Jules J. Sarrazin, of New Orleans, Louisiana.

Fig. 497.

Fig. 497. A brush with the long tuft of bristles on the end showing how this tuft prevents the shorter near-by bristles from touching the teeth, also how the long bristles fold back in positions where there is not ample room.

refused to cut away had been removed by absorption. The new line of gingivæ was hard and firm in every part, and smooth enough to be easily cleanable.

Clearly my patient had the best of me, or to put it differently, she succeeded in retaining the tooth in spite of me. She said that in the fifteen months that she had been away she had not missed the full cleaning process three times per day, and that the third molar always had two extra syringefuls of water. She was delighted with the result.

The gingivæ were very short. The whole circle of the gingival line could be seen upon many of the teeth, and some part of it on all of them. As had been noted at first, the lips covered the teeth so well that the loss of the gingivæ was not noticeable. Her teeth made an excellent showing, both in talking and in laughing. Her bite was strong enough to manage a beefsteak or other of the ordinary foods.

Artificial cleaning unnecessary for some persons. There is much variation in the requirements in artificial cleaning among different people. Some persons under forty years need no artificial cleaning. I have examined a few such persons within an hour after a meal, and although a tooth-brush had not been used, their mouths were as clean as those of the most careful and fastidious patients. It seemed that debris which remained after meals was dissolved — digested by their saliva — and removed completely in a short time.

One rather notable person whom I frequently had under observation during the latter part of her life, had never owned a tooth-brush. It was her continuous habit after meals to take a glass of water, and filling her mouth, force this through between and among the teeth, and cast it out. As I often saw her do this, she used up the full glass of water in mouthful after mouthful, and there the cleaning ended. She never had a decayed tooth, she never had a crust of calculus, and at seventy-six, when she died of pneumonia, her gingivæ were as full and complete, and rose as high upon the crowns of the teeth as was normal for the girl eighteen years of age.

A few patients of this class came regularly several times a year for examination. If more of those people who need but little attention would do this, many cases of disease of the peridental membrane would be prevented. It is important to remember that persons who are immune to caries may have deposits of calculus, and because of the preëxisting sense of security which the normal cleanliness had engendered, it will be difficult

to bring them to take sufficient care when artificial cleaning becomes necessary. These are usually very difficult cases to manage. It will often require all of the influence that can be brought to bear, to make them realize the necessity of frequent and thorough brushing.

Irregularities of the gingivæ. Scars of the gingivæ result from previous injuries, which have caused changes from the normal form. These require extra care in brushing, which must be varied to meet the particular case. There may have been a shallow pocket formed in the attachment of the peridental membrane of a certain tooth. This may have recovered, leaving a notch in the regular line of the crest of the free gingivæ, in which debris will lodge and be difficult to remove. In another case, the crest of the gingivæ may have become thickened, forming a shelf where it lies against the tooth, which tends to the collection of debris. A still more serious condition arises from the shortening of a septum here and there, which calls for special care in the cleaning of these interproximal spaces. In old age the whole of the free gingivæ sometimes become short and much thickened at their margins, and the septal gingivæ no longer fill the spaces between the teeth.

All such injuries tend especially to increase the lodgment of deposits, both of food debris and calculus, and in this way endanger the health of the parts. This danger occurs mostly because of the tendency of such defects to cause lodgments. Most of the irregularities of position of the teeth present their own particular difficulties to the cleaning process. The cleaning must be varied to meet these conditions. If the gingivæ are blunted, they will be gradually thinned by regular brushing with proper motions.

Cleaning artificial dentures.

Artificial dentures or bridges, either fixed or removable pieces, should receive more attention in the matter of cleanliness than the natural teeth. This is necessary to the full and comfortable use of such appliances. Neglect is sure to bring serious inflammation sooner or later.

The epithelium of the mouth, which is fitted for the friction it receives in chewing food, is continually giving off dead cells from its surface and renewing them by fresh growth. If a portion of this mucous membrane is constantly covered by a denture, which fits so well that food debris does not get under it, the dead cells will remain and within a day or so will cause the

entire surface of the mucous membrane under the denture to be slightly or considerably whitened. If these dead cells are not brushed away, they will soon decompose and render foul the whole surface of the denture in contact with the tissue. It often happens that more or less food debris will work in under some dentures, and this will decompose, causing inflammation of the mucous membrane. It is, therefore, a necessity to the comfort and full use of the denture that the cleaning of it and the mouth be done at regular intervals, the same as the cleaning of the natural teeth.

The denture should be placed under the hot-water faucet, if running water can be used, while the mouth and the natural teeth, if some remain in the mouth, are cleaned. The mucous membrane, over which the plate fits, should receive a very thorough cleaning with a brush — one having bristles rather softer than those used for cleaning natural teeth. This will remove all deposits and dead epithelium which may have collected under the denture, and give to the tissues a sense of comfort.

The denture should be examined, by touching it here and there with the fingers. It will soon be recognized that the mucus, which gave it a slippery feeling when it was removed from the mouth, has been washed away by the water. If some little calculus has collected here and there, this will remain and such spots will feel greasy to the fingers. This deposit of soft calculus can not be removed by running water, but is easily removed by vigorous brushing. For this purpose, a small hand brush, with the bristles thickly set, should be used. (See Figure 500.) Nothing besides the brush and water is needed for cleaning the plate, though there is no objection to using a little soap. The brushing should be continued until the plate is perfectly clean in every part. The movements of the brush should include those necessary to bring the bristles through the embrasures between the teeth on both the buccal and lingual sides. That part of the palatal surface which lies next to the tongue, as well as the surface which comes in contact with the mucous membrane, should be made thoroughly clean.

Then that part of the plate which covers the residual alveolar ridge should be brushed with the special brush, designed for the purpose, and care should be taken to reach every part of this surface. (See Figure 499.) It is not sufficient to simply place the brush in the groove and brush around the groove. The angle at which the brush is held must be changed so that every part will be cleaned. Some regular order should be followed so that

every part of the plate will receive sufficient brushing to render it clean.

If there is calculus coming into the mouth, more or less of it will be deposited somewhere on the plate, and if the cleaning is neglected, this deposit will become so hard that it can not be removed by the brush. Whereas when the deposit is fresh, having accumulated between meals or over night, as the longest period between cleaning, it will be so soft as to be easily removed by brushing. Therefore, if one neglects the cleaning, the plate will soon be in such condition that it can not be well cleaned with the brush. Then it will cause more and more inflammation of the mucous membrane until it is again polished. Persons will soon learn that the deposits of calculus occur in the same places time after time. These are especially the buccal surfaces of upper plates about the molars and the lingual surfaces of lower plates in the incisor region.

Neglect of cleaning will lessen the comfort and usefulness of the plate, and in time the mucous membranes, over which it fits, may become so inflamed that it can not be used with the vigor which is necessary to the proper chewing of food. If the use of a foul plate is persisted in, the condition of the mucous membrane may become such as to prevent its use entirely, or render such usage very uncomfortable and inefficient. I have seen mouths so sore that the plate could not be used at all in the chewing of food. It is not my intention, however, to go into the consideration of the diseases of the mucous membrane induced in this way.

The form of the surface next to the lips and cheeks should be of even fulness, and the embrasures between the teeth should be filled out so full that food crushed between the teeth will run through them with such force as to tend to keep them clean. They will then be of the best form to facilitate cleaning with the brush. There should be no irregularities of the surface in any part of the denture, and the polish should be as perfect as it can be made. This matter of polish is of great importance in preventing the catching of deposits of calculus by the plate. Dentures which have been worn for some time will receive the first deposits in low places upon the surface, over which food will pass without rubbing.

The dentist should study this point with the greatest care and so form the surface of the plate that it will not be liable to catch and hold debris of any kind and will not readily catch salivary calculus. It is quite possible to so construct and polish a

Fig. 498.

Fig. 498. The rubber bulb syringe for patients to use in washing interproximal and subgingival spaces. The nozzle has a 1.5 mm. hole in the end. It is carried along the crests of the gingivæ, the end touching the enamel close to the gingivæ, while held at such an angle that the water will strike the enamel and be directed into the subgingival spaces. This is a very effective means of keeping the gingivæ in the best condition, and of cleansing the interproximal spaces, particularly in cases in which the septal gingivæ have receded a little.

Fig. 499. Fig. 500.

Figs. 499, 500. Brushes for artificial dentures, actual size. The brush shown in Figure 499 is designed especially for brushing the part of the plate which covers the residual alveolar ridge. For plates with high rims the tuft on end must be enough longer to easily reach the portion which covers the alveolar ridge. The larger brush, Figure 500, may be used for the rest of the denture. This is a small hand brush.

plate as to avoid any calculus whatever from adhering to it. Perhaps it is not possible to maintain this in usage. Its maintenance will require repolishing now and then, but this can be easily done, and thus keep the plate in a condition so that calculus will not adhere, as has been noted in the articles describing conditions of fresh deposits of salivary calculus.

I remember well one patient, the case occurring a number of years ago, for whom I had extracted all of the teeth when she was about thirty years old, on account of disease induced by persistent deposits of calculus, and had made artificial dentures. I requested her to return several times for examination. I instructed her carefully in the matter of cleaning both her mouth and the dentures, and she seemed to fully understand what she should do. I heard nothing from her for about six months, when she came in complaining that her mouth was sore under the plates and that the plates had become rough. Upon examination I found heavy deposits of calculus upon them and the mucous membranes were sore wherever the dentures fitted against them, as would naturally be the case from the condition in which I found the dentures as to cleanliness. I called her attention to this and told her that she had not kept the plates properly cleaned. I cleaned and polished them anew. Then I sat down with her and talked to her about the necessity of cleaning artificial teeth and went over in detail the whole plan of cleaning and the results that were likely to occur if my directions were not followed. It was evident that she was very much offended, but I finished what I had to say. She went away without a word. I never expected to see her again. About six months after that she came in and asked me to examine her plates and her mouth. I looked them over very carefully and simply said "excellent." She told me that the principal object of her visit was to thank me for what I had done for her when she last visited me.

Cleaning bridges.

Persons for whom fixed bridges have been placed are likely to find discomfort in the lodgment of food debris about such pieces, unless they have been made with due care to render the cleaning of them easy. The exposed parts of such pieces may be cleaned by the ordinary brush used in cleaning natural teeth, but that portion directed toward the mucous membrane can not be well cleaned in this way, and other means should be devised by the dentist for the particular case. No part of a bridge should press on the mucous membrane; it should be supported wholly

by its abutments. It should not come so close to the mucous membrane but that a piece of thin tape may be passed between the two and used to rub over the part of the appliance that can not be reached by the brush. For this to be effective, the surface of the dummies toward the tissues should be convex from buccal to lingual, rather than concave as many are made, especially in the portion of the surface toward the lingual. This rubbing by a simple cotton tape, carrying no abrasive whatever, followed by the syringe, will complete the cleaning.

If the cleaning is regularly done after each meal, the bridge should be kept in excellent condition. If the cleaning is not regularly done, inflammations of the mucous membrane about the bridge and under it are very likely to occur. Furthermore the soft tissues about the teeth and roots, to which the bridge is attached, are liable to inflammation, which may result in the loss of both the teeth and the bridge. These difficulties make the continued use of the bridge after many years very doubtful. Although originally constructed so as to facilitate cleaning, changes may occur to render the cleaning more and more difficult until disease is induced about the roots supporting the bridge.

Bridges require more care than any other of the artificial appliances placed in the mouth. Similar care must be exercised in the construction of the bridge; it should be made with the utmost precaution that every part be thoroughly cleanable and present sufficient space about the mucous membranes to give free access to the natural movements of the fluids of the mouth. This and the continuous cleaning are essential to the best results.

Much harm is being done by bridges which are neglected or can not be properly cleaned. They become a menace to health by the inflammations which they cause and become harbors for micro-organisms. The dentist should, whenever a bridge is set, instruct the patient in the means of cleaning it. At subsequent visits he should note whether or not the cleaning is being done properly.

EXAMINATIONS OF THE MOUTH

By ARTHUR D. BLACK, M. D., D. D. S.

ILLUSTRATIONS: FIGURES 501-507.

THE importance of very painstaking and thorough examinations of the mouth has been emphasized in the consideration of each subject presented. The service of the dentist will increase in effectiveness toward prevention as he learns to recognize the beginnings of disease. In proportion as inflammations of the gingivæ are recognized and treated early, will cases of chronic suppurative pericementitis be prevented; likewise, as decays are discovered early, will cases of pulp death and alveolar abscess be diminished. No elaboration of these statements seems necessary to one who has read this book.

The subject of mouth examinations is included here for the purpose of presenting a systematic plan of examining and recording all conditions which should be noted. The record of an examination is of almost as much importance as the examination itself, by reason of the fact that the habit of recording necessitates a clear mental picture of the condition to be recorded. This begets keener observation and develops the faculty of logical thought and proper deductions, as applied to the conditions presenting. The man who develops the habit of making accurate records will soon learn to see as much more in mouth examinations as does the trained microscopist as compared with the beginner. The record will often be found of much value in the later treatment of cases, and the gradual accumulation of records should eventually be the basis of tabulated reports which would prove to be the best guide for future practice.

While this book is devoted to the diseases of the gingivæ, peridental membrane and dental pulp, these conditions are so closely related to other procedures that it seems desirable to present a plan for a complete mouth examination. This plan is one which has been followed in its principal features by the writer for fifteen years. When once understood, its application is much more simple than the rather extended description of the details might indicate. Its practicability has been quite thoroughly tried out by a number of operators.

Persons who apply to the dentist may be placed in three groups: 1. Those who present at more or less definite intervals for routine examinations, usually without any particular complaint. 2. Those who come because of some particular condition of pain or discomfort, which may require that the examination be directed to this condition, and contraindicates a complete mouth examination at the time. 3. Those who are suffering from some secondary infection for which a mouth focus is sought. We need consider only the routine examination for the first group, as it will include the conditions presenting under the second and third groups.

As a part of every examination the dentist should have the best possible opinion of the patient's general physical condition. This is important, not only in connection with the management of regular patients who come for routine examinations at stated intervals, but also for those who may present for the first time while suffering, or who may be referred by the physician on account of some systemic effect of a local focus. While it is without the sphere of the dentist to make a full physical examination, he may by such observation and inquiry as the circumstances will permit, gain much information which will be of service. When there are mouth infections which are evidently of long standing, and particularly if the patient seems not to be in robust health, the dentist should refer the patient to a physician for a thorough physical examination. The patient's general manner, alertness in movements and conversation, the color of the skin, the facial expression, etc., will usually give the dentist sufficient information to guide him in the conduct of his operations. It is of course understood that the pulse and temperature will be taken in all cases in which there is an indication for so doing. The point to be emphasized here is that the dentist should cultivate his powers of observation to enable him to determine the best course to pursue in the management of patients, in order that his service may be most effective.

ROUTINE MOUTH EXAMINATION.

A complete examination of both the soft and hard tissues of the mouth should be made at regular intervals, the frequency to be determined for each patient. As a general statement, it might be said that for the majority of children under ten years of age, the examinations should be every three months; for most

persons from ten to twenty years of age, three times or twice a year; for persons past twenty, twice a year.

Dentists who will adopt a reliable plan for notifying patients, should offer to take the responsibility of sending notices for such examinations. Unless a plan is followed by which there will be little likelihood of failure to send notices at the proper times, the responsibility had better be left with patients.

A good plan is to have a memorandum column in the regular appointment book, so that, as a series of operations for each patient is finished, the name may be entered in this column under the date on which the next notice for examination is to be sent. There is little opportunity to overlook such a memorandum, as it will be seen alongside the regular appointments when the time arrives. This plan requires that the book for the next year be ready six months or more in advance, or one may set aside a few pages in the back of the book of the current year — one for each month — and make the entries there, subsequently transferring them to the proper places in the book for the new year.

A card system may be used for this purpose. For this, one should have monthly guide cards, and the cards for patients who are to be notified may be placed back of the guide for the month when the next notice is to be sent. The same guide cards and the same patients' cards may be used indefinitely. The date should be entered on the patient's card whenever a notice is sent. Under either plan it will be found most convenient to send out all of the notices for each month on a single day, or possibly on two days — the 1st and the 15th.

Some patients will prefer to take upon themselves the responsibility of their return, and the suggestion that the dentist notify them will not be acceptable. The dentist should therefore do no more than to offer to make a memorandum and send such a notice if the patient desires him to do so. When notices are sent it is well to state that the patient may cancel the appointment if the time set is not convenient. The patient should be impressed with the fact that the arrangement is made primarily for the best care of his mouth, and not as an economic procedure for the benefit of the dentist.

In making a routine examination the dentist should have in mind the following conditions:

1. The occlusion.
2. General condition of the teeth.

3. General appearance of the entire mucous membrane of the mouth.

4. Condition of mouth as to cleanliness.

5. Inflammations caused by deposits of salivary calculus.

6. Gingivitis caused by deposits of serumal calculus.

7. Injuries to gingivæ caused by open contacts or bad contacts; by imperfect margins of fillings or crowns, etc.; or by abuse of the tissues in dental operations.

8. Injuries caused by misuse of toothpicks, rubber bands, brushes, etc.

9. Detachments of the peridental membrane.

10. Chronic alveolar abscess.

11. Hyperemic, inflamed or dead pulps.

12. Caries.

In the actual examination a definite routine should be followed which will include all of the above, although it is not necessary to make a separate survey of the mouth for each item mentioned. The plan presented herewith will be found to be very satisfactory. It is not important that this particular plan be followed. Any other which covers the field will do as well. It is of the utmost importance, however, that each dentist train himself into the habit of following a regular plan in order that nothing may be overlooked. Each condition should be recorded as the examination proceeds. A detailed statement of a simple plan of making this record will be given.

Instruments for routine examination.

The following instruments should be on the operating tray, or conveniently at hand, for this examination:

Mouth mirror.

Three explorers, one almost straight, and a pair of right and left curved instruments.

Pair of peridental membrane explorers.

Cotton pliers.

Cotton.

Silk floss.

Air syringe.

Water syringe, and warm water.

In addition to the above, a mechanical separator will often be required for examination of proximal surfaces. Also a sharp steel probe should be used to explore practically every sinus, and occasionally a soft, blunt silver probe will be needed.

The examination should consist of a general survey of the entire mouth, followed by a more critical inspection of the teeth and adjacent tissues of the lower jaw, then a similar inspection of the upper. For the general survey the chair should be tipped about half-way back; for the inspection of the lower teeth and soft tissues it should be upright; for the uppers it should be tipped far back.

General survey.

For the general survey the lips should be retracted so as to give the best possible view, first of one side of the mouth, then of the other. The patient may then be asked to close the teeth so that the occlusion may be noted. The general condition of the teeth, as to atrophy or hypoplasia, erosion or abrasion, should be observed; also an estimate should be made of the extent and condition of previous dental operations. The entire mucous membrane should be carefully inspected, noting points of swelling, abnormal redness, sinus openings, or any abnormal conditions. The condition of the mouth and teeth as to cleanliness should be carefully observed at this time; also the general condition as to deposits of salivary calculus. The mouth mirror should be used for the examination of the lingual surfaces.

In this survey a good general idea will be had of the conditions presenting in the particular mouth. The number of fillings, the number of teeth missing, the extent of decays which may be observed, the general condition of previous operations, the cleanliness or lack of cleanliness, together with the age, the apparent general manner, physical and nervous condition, should enable the dentist to form a good opinion, not only of the condition of the investing tissues and the susceptibility of the patient to caries, but of the problems and difficulties to be met both in the operations to be performed and in the direction and training of the patient in mouth hygiene.

Critical examination of the teeth and investing tissues.

A regular routine should be followed for each arch, and the examination of the lower arch should be completed before changing the position of the chair for the examination of the upper arch. As good a plan as any will be to begin with the third molar on the left side and pass gradually around the arch to the right third molar.

The investing tissues should be examined first, beginning with the crests of the gingivæ. Recessions and inflammations of

Chart for Examinations of the Mouth

Routine to Be Followed.

General Survey. Chair tipped half way back.

Conditions.	Suggestions for Entries on Record.
1. Occlusion.	Normal, or class (Angle), or indicate particular teeth with arrows.
2. General condition of teeth as to decay.	Good. Immune. Very susceptible. Previous fillings good, etc.
3. General condition of mucous membrane.	Good. 17. 18. 19.
4. General condition of mouth; cleanliness.	Good. 70 or subdivisions.

Critical Examination of Teeth and Investing Tissues. Chair upright for lower jaw, far back for upper jaw.

5. Labial, buccal and lingual gingivæ.	Recessions, 17. Salivary calculus, 18. Serumal calculus, 19. Imperfect margins of fillings, crowns, etc., 40 and subdivisions. Abuse of tissues in operations, 50 and subdivisions. Lack of cleanliness, 60 and subdivisions. Errors in cleaning, 70 and subdivisions.
6. Septal gingivæ.	Lack of contact, 20 and subdivisions. Improper contact, 30 and subdivisions. Imperfect margins of fillings, crowns, etc., 40 and subdivisions. Abuse of tissues in operations, 50 and subdivisions. Lack of cleanliness, 60 and subdivisions. Errors in cleaning, 70 and subdivisions.
7. Detachments of peridental membrane.	Enter depth in millimeters, 1-15 (Table 2), or use 1, 2, 3, as indicating shallow, medium and deep, without actual measurement.
8. Digital examination, absorptions of bone, presence of pus.	Write word *absorption* or *pus*.
9. Exploration, sinuses.	Write word *abscess*, or C (see Table 2).
10. Condition of pulps.	T, or subdivisions in Table 2.
11. Order, radiographs.	Small x above tooth.
12. Caries, atrophy, abrasion, erosion.	Symbols for caries. Mark location of others with red ink, enter word for condition.

Note.— This chart has been prepared as a guide to be followed in mouth examinations. After each item, memoranda are given for the entries to be made in making the record. If copies of this chart and the accompanying tables are placed where they may be conveniently referred to and followed for a time in making mouth examinations, one will soon become familiar with most of the details. It will be observed that there are two tables of numbers, letters and symbols; one enabling the operator to record much closer detail than the other. One may follow the more simple plan of Table No. 1 for a time, until he is familiar with it and then change to Table No. 2. In doing this there will be no confliction. In recording inflammations of the gingivæ, for example, the numbers in Table No. 2 give a more exact statement of causes than do the numbers in Table No. 1. See illustrations 502 to 507, which show a sufficient variety of entries to make the plan clear.

Markings to be Used in Recording Examinations of the Mouth

SEE DESCRIPTIONS OF FIGURES 501 TO 507.

Table No. 1—Partial Detail.

Conditions of the Investing Tissues.

16. Open contact, no inflammation.

17. Recession of gingiva, cause not apparent.

Inflammation, Due to

18. Salivary calculus deposits.

19. Serumal calculus deposits.

20. Lack of contact of teeth.

30. Improper contact of teeth.

40. Deviations from normal contour — bad margins of fillings, crowns, etc.

50. Abuse in previous dental operations.

60. Lack of cleanliness.

70. Injuries in mouth hygiene technic.

Depth of pockets, 1, 2 or 3, indicating shallow, medium or deep.

Conditions of Pulp, Etc.

T Treatment required, without reference to condition.

Caries, Etc., Operations.

○ Pit or fissure decay.

() Proximal decays, mesial and distal.

⊃ Gingival third decays, labial or buccal and lingual.

/ Missing tooth, previously extracted or unerupted.

X To be extracted.

O To be crowned.

= Dummy.

| Partial denture. Line through each tooth and these lines joined.

Full denture, write same on diagram.

Table No. 2—Complete Detail.

Conditions of the Investing Tissues.

16. Open contact, no inflammation.

17. Recession of gingiva, cause not apparent.

Inflammation, Due to

18. Salivary calculus deposits.

19. Serumal calculus deposits.

Lack of Contact of Teeth.

21. Separations following extractions.

22. Abnormalities of occlusion.

23. Uneven occlusal wear.

24. Weak contact.

25. Proximal decays.

26. Fillings or crowns.

27. Loss in width of neighboring space.

28. Detachment of trans-septal fibers.

Improper Contact of Teeth.

31. Abnormal forms of teeth.

32. Malpositions of teeth.

33. Interproximal wear.

34. Improperly finished fillings and crowns.

Deviations from Normal Contour.

41. Sharp edges of cavities.

42. Imperfect margins of fillings.

43. Imperfect margins of crowns.

Abuse in Dental Operations.

51. Injuries with ligatures.

52. Injuries with finishing instruments and tapes.

53. Failures to remove ligatures and pieces of rubber.

Lack of Cleanliness.

61. Lack of natural cleaning in mastication.

62. Lack of artificial cleaning.

Errors in Cleaning Operations.

71. Misuse of toothpicks.

72. Misuse of rubber bands, silk floss.

73. Injuries with tooth-brush.

Conditions of Investing Tissues — Continued.

Detachments of Peridental Membrane.

1 to 15. Depths of pockets in millimeters, measured with special graduated explorers. (See Figure 267.)

Conditions of Pulp, Etc.

T Treatment required without reference to condition.

N Normal.

H Hyperemia.

E Exposed and alive.

D Dead.

R Previous root filling.

A Acute alveolar abscess.

C Chronic alveolar abscess.

Radiographs.

x Small cross above each tooth roots and alveolus of which is to be shown in radiographs. Red ink preferred for this.

Caries, Etc., Operations.

○ Pit and fissure decay.

() Proximal decays, mesial and distal.

⊃ Gingival third decays, labial or buccal and lingual.

/ Missing tooth, previously extracted or unerupted.

X To be extracted.

O To be crowned.

= Dummy.

| Partial denture. Line through each tooth, and these lines joined.

Full denture. Write same on diagram.

Atrophy, abrasion, erosion to be marked with red ink and name on bottom line to indicate condition.

the buccal and labial, then of the lingual gingivæ, should be noted; also deposits of salivary calculus, improper margins of fillings and crowns, and other causes of the different areas of inflammation. Then the septal gingivæ should be carefully examined for areas of inflammation. The cause of each area of gingivitis should be determined, the search being based upon a knowledge of the wide variety of conditions presenting. This will include an examination of the contact in each case in which a septal gingiva is inflamed. It may lead to inquiry into the patient's habits of cleaning, the misuse of toothpicks or silk floss, the kind of tooth-brush, the motions made with the brush, etc. All inflammations of the gingivæ call for an instrumental examination of the subgingival spaces for deposits of serumal calculus. Sharp pull scalers will usually be preferred to the peridental membrane explorers for this purpose. For adults, the subgingival spaces to the lingual of the upper incisors should always be examined for these deposits.

The peridental membrane explorers should next be employed to determine whether or not there have been detachments of the membrane from the cementum. Such instruments may be carried into the subgingival spaces until they meet the attachment to the cementum, and may then be passed around the tooth, noting any deviations from the normal line. The depth and positions of pockets may be noted.

Absorptions of bone of the alveolar process on the labial and buccal sides of the arch, in cases of chronic pericementitis, may be noted by digital examination. The finger, passed along the mucous membrane, will detect positions where the bone is missing, and something of the contour of the root may often be made out. On account of the thickness of the investing tissues, absorptions to the lingual of the arch can not so often be detected. In cases of chronic abscess, absorptions which have involved the bone to its surface may be discovered; or enlargements resulting from the building of new bone will be found.

As a part of this digital examination, pressure may be made on the sides of the arch to determine whether or not pus is present in a pocket, by forcing it out at the gingival line. Teeth may be tested as to their firmness in their respective sockets.

If there is a sinus, it should be explored to find its source, the sharp steel probe being generally used for this purpose. If the sinus is of some length and tortuous, the blunt silver probe will be better to follow it. As has been emphasized, it is of the

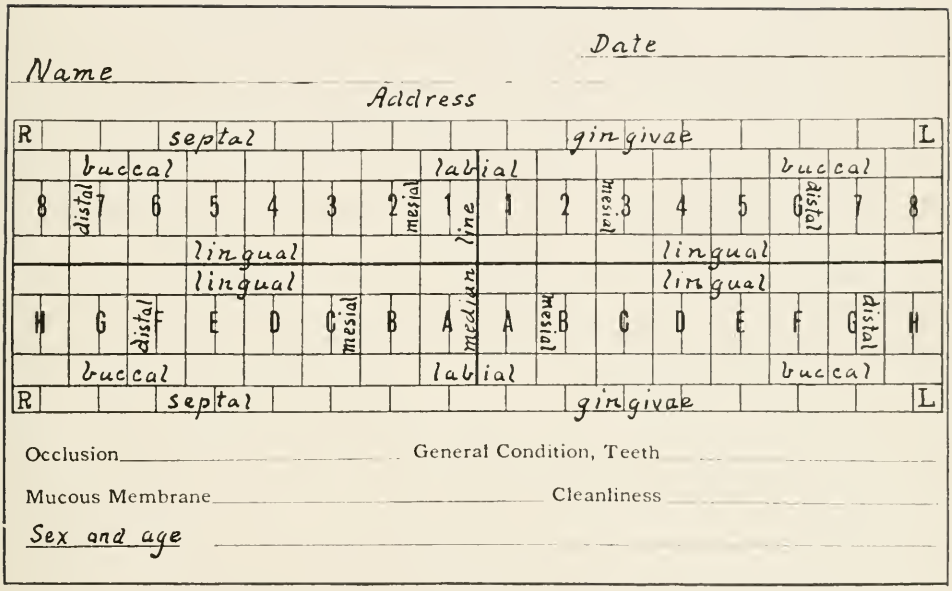

FIG. 501.

Fig. 501. The Examination Card. Size 3 by 5 inches. The figures represent the upper teeth on either side of the mouth; 1, central incisor; 2, lateral incisor; 3, cuspid, etc. The letters represent the lower teeth on either side. The patient is supposed to be facing the operator and the right and left sides of the mouth are indicated by R and L in the corners of the diagram. The occlusal surfaces and incisal edges of the teeth are supposed to be directed toward the operator, therefore the buccal surfaces of the upper teeth would be up, and the buccal surfaces of the lower teeth would be down. It will be noticed that there is a rectangular space on either side and one above and one below each figure or letter; numbers may be placed in these spaces to indicate the condition of the investing tissues on the mesial, buccal, distal or lingual sides of each tooth. There is another row of rectangular spaces at the top, and one at the bottom of the diagram; numbers may be placed in these to indicate the condition of the septal tissue and of the contact. See markings on other illustrations.

This plan of using figures and letters, instead of diagrams, to designate the teeth, enables one to transfer the record for a single tooth from daybook to record or ledger. For example, in Figure 502, a cavity is marked in the mesial surface of the upper right first molar; this can be written alone as 6) , the line indicating that the tooth is on the right side. The mesial of the lower first molar on the left, in the same illustration, would be written alone (F.

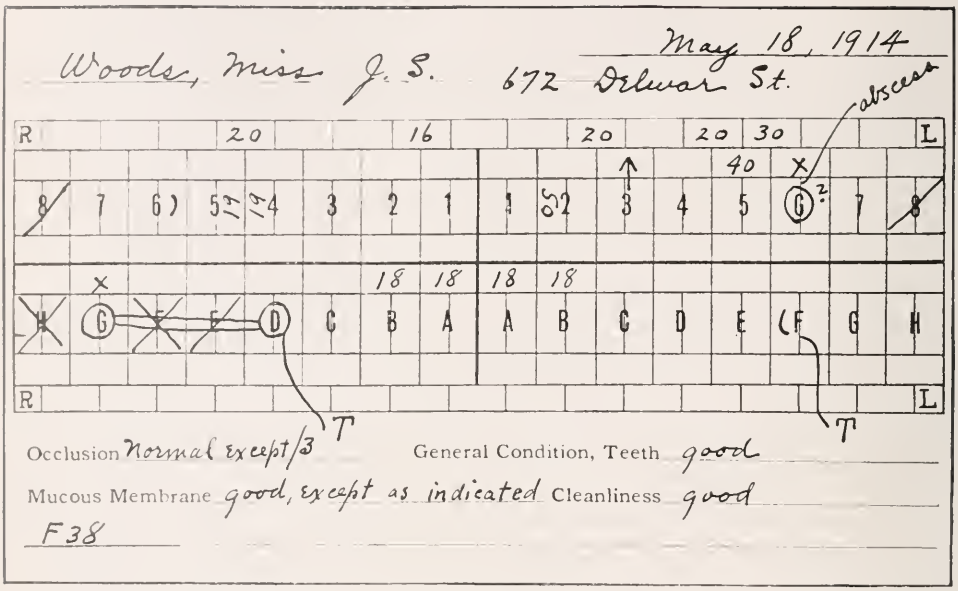

Fig. 502, A.

Fig. 502, A. Record of Examination. (Marks from Table No. 1.) Patient, woman of thirty-eight years. General condition of teeth as to susceptibility to caries, good; general condition of mucous membranes, good. Care of mouth, good.

In the upper arch, both third molars are missing; there is a cavity in the mesial surface of the right first molar, there is an open contact between the bicuspids, the septal gingiva being inflamed (20), and there are deposits of serumal calculus (19) on the proximal surfaces of these teeth. There is an open contact between the right central and lateral, but the septal tissue is not inflamed (16). There is an inflammation of the gingiva to the mesial of the left lateral on account of some abuse in operating (50). The left cuspid is to the labial of the line of the arch as indicated by the arrow, and there is an inflammation of the septal gingiva between the cuspid and lateral on account of lack of contact of these teeth (20). There is an inflammation of the gingiva to the buccal of the second bicuspid due to a bad margin of a filling or crown (40). There is an inflammation of the septal gingiva to the mesial of this tooth on account of an open contact (20), and to the distal of it on account of an improper contact (30). The first molar has an abscess, and the little cross shows that a radiograph is to be made. It is marked to be crowned, the question mark indicating that this operation is in doubt on account of the abscess.

In the lower arch, there are deposits of salivary calculus (18) to the lingual of the lower incisors, there is a mesial cavity in the lower left first molar, and the pulp requires treatment (T). The right second bicuspid is missing and the first and third molars are to be extracted. A bridge is to be placed from the second molar to the first bicuspid. The first bicuspid is marked for pulp treatment (T). The second molar is marked for a radiograph.

Compare with description of Figure 502, B.

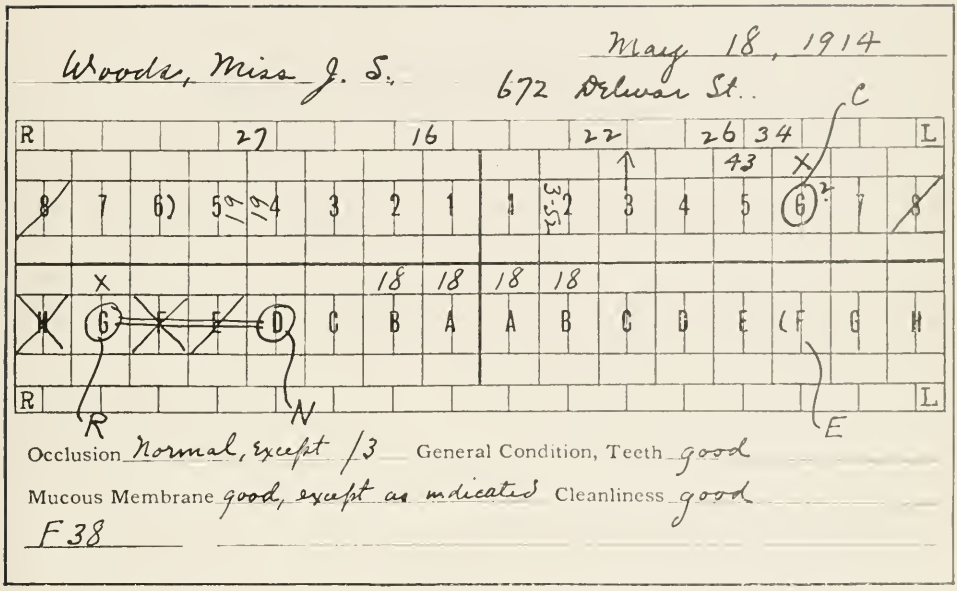

Fig. 502, B.

Fig. 502, B. Record of Examination. (Marks from Table No. 2.) This is the same case as in Figure 502, A. The use of the more complete detail in recording will be understood from a comparison of the two. The differences are as follows: In the upper arch, in A, an open contact is marked between the right bicuspids (20). In B, the number 27 shows the open contact to be due to a flat proximal filling or cavity in a neighboring tooth. Apparently these teeth have separated on account of the cavity in the mesial surface of the first molar. In A, an injury in operating is recorded on the mesial surface of the left lateral (50). In B, the number 52 shows this to be due to an injury by a finishing instrument, and there is a pocket 3 mm. deep. In A, an open contact is recorded between the left lateral and cuspid (20). In B, the number 22 shows this to be due to an abnormality of occlusion. In A, another open contact is recorded between the bicuspids (20). In B, the number 26 shows this to be due to lack of contact of a filling or crown. In A, there is an inflammation to the buccal of the first molar, due to a deviation from the normal smooth contour (40). In B, the number 43 shows this to be due to the margin of a crown. In A, there is recorded an inflammation of the septal gingiva between the second bicuspid and first molar, due to an improper contact (30). In B, the number 31 shows this to be due to an improperly formed filling or crown.

In the lower jaw, in A, the pulp of the lower left first molar is marked for treatment (T). In B, it is shown that this pulp is exposed and alive (E). On the lower right side, the letter N indicates that a normal pulp is to be removed from the first bicuspid and the R that the root canals of the second molar have been filled by a previous operator. There is apparent no sign of an abscess; the radiograph has been ordered to learn the condition of the canals and of the bone about the apex.

It requires only a little more time to become familiar with Table No. 2, and one is then able to make better records without occupying more time in marking the cards.

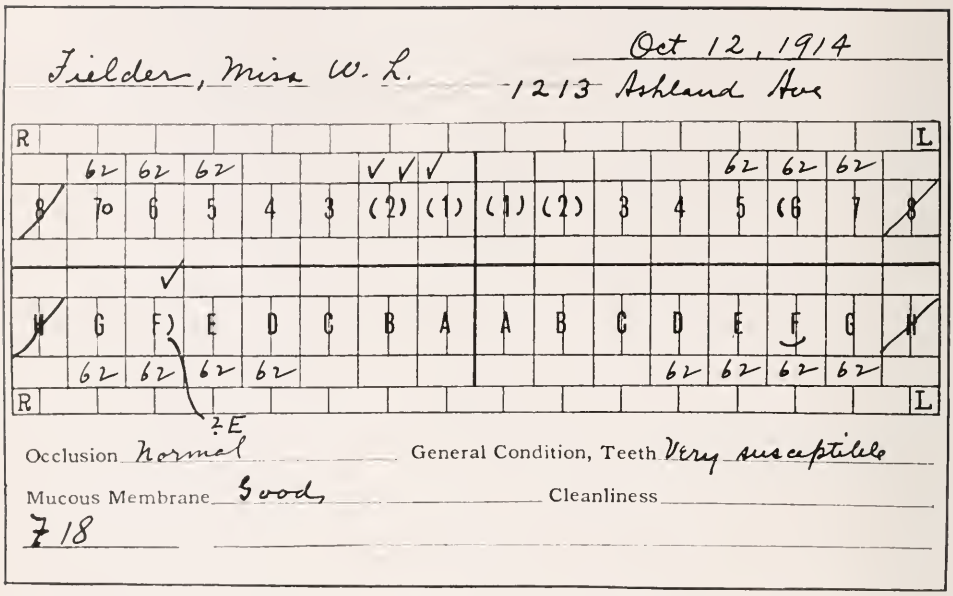

FIG. 503.

Fig. 503. Record of Examination. (Marks from Table No. 2.) Patient, girl of eighteen years. Occlusion normal. Susceptibility high to caries. Mucous membranes, good. The number 62 indicates improper artificial cleaning of most of the bicuspids and molars, and there is one gingival third decay in the lower left first molar because of this. Patient's attention is called to the condition at the time of the examination and is told how to brush these teeth and the kind of brush to use. When she returns for subsequent operations, the card reminds the dentist of this, and her care is criticized from time to time. Three or four months later, when patient returns for another examination, the card again reminds the dentist to criticize the care of these areas.

There are mesial and distal cavities in the four upper incisors, an occlusal cavity in the upper right second molar, a mesial cavity in the upper left first molar, and a mesial cavity in the lower right first molar. There was some question at the time of the examination as to whether or not the pulp of this tooth might be exposed, as indicated by the question mark before the letter E.

The check marks indicate that at the time this card was photographed, the filling had been placed in the lower right first molar, also that three fillings had been placed in the upper incisors. By checking each operation as it is recorded on the permanent record, the operator has before him, at each return of patient, an exact memorandum of what has been done and what remains to be done.

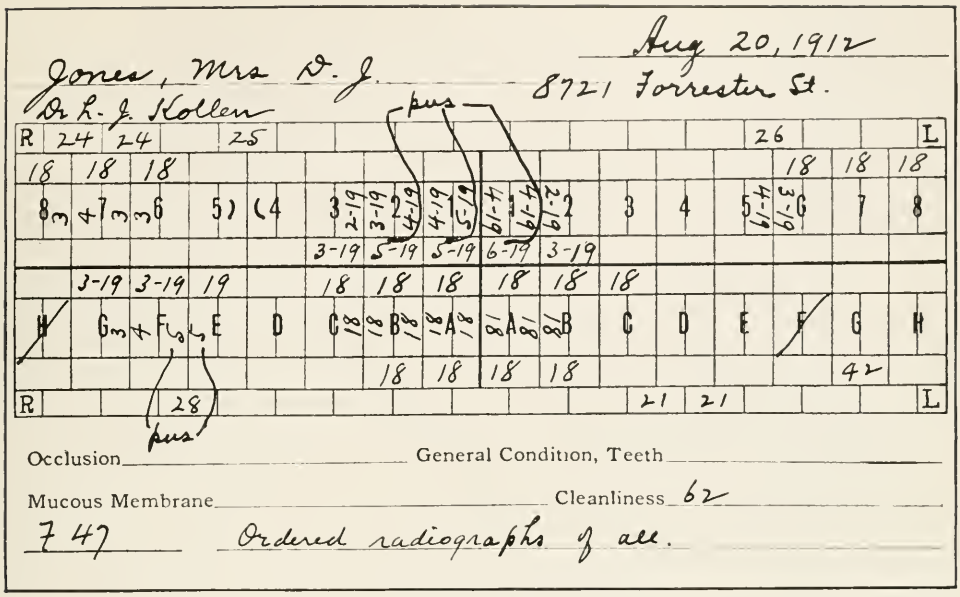

FIG. 504.

Fig. 504. Record of Examination. (Marks from Table No. 2.) Patient, woman of forty-seven years. Referred by Dr. L. J. Kollen, with request for report on mouth as probable focus of infection. The record shows a mouth not well cared for by the patient (62), with deposits of salivary calculus (18) on the buccal of the upper molars, also on the lingual and proximal surfaces of the lower six front teeth, and on the labial surfaces of the four incisors.

In the upper arch, on the right side, the contacts between the molars are weak (24), permitting food to be forced through. There are pockets 3 and 4 mm. deep on the proximal surfaces of these teeth. The septal gingiva between the bicuspids is inflamed because of the two proximal cavities (25). There are pockets of various depths — from 2 to 6 mm. on the lingual and proximal surfaces of the incisors and right cuspid. Deposits of serumal calculus are indicated on all of these surfaces (19). Pus was discovered to the lingual of the right lateral and both centrals. There is inflammation between the upper left second bicuspid and first molar, due to a filling or crown which failed to make contact (26). There are pockets 4 and 3 mm. deep, and deposits of serumal calculus (19).

In the lower jaw, in addition to the deposits of salivary calculus, there are pockets on the lingual surfaces of the first and second molars 3 mm. deep, with deposits of serumal calculus (19). There is a deposit on the enamel of the lingual subgingival space of the second bicuspid (19), but no pocket. There are pockets on the proximal surfaces of the bicuspids and molars, 3, 4 and 5 mm. deep, but no deposits. Pus was found in two of these. The contact between the second bicuspid and first molar is open as a result of the cutting off of the trans-septal fibers which normally pass from tooth to tooth (28).

On the lower left side the first molar is missing and the bicuspids have moved distally, opening the contacts and permitting an inflammation of the septal tissues (21). A gingival third filling in the buccal surface of the second molar has a bad margin and the gingiva is inflamed (42).

Radiographs were ordered for the entire mouth.

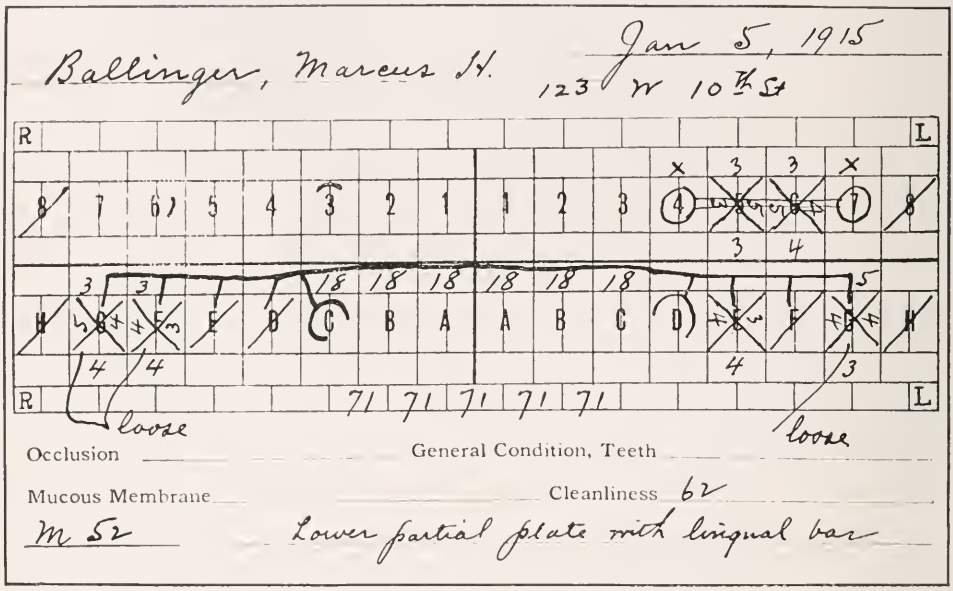

FIG. 505.

Fig. 505. Record of Examination. (Marks from Table No. 2.) Patient, man of fifty-two years. There is a general lack of care (62). In the upper arch there is a mesial decay in the right first molar and a gingival third decay in the labial surface of the cuspid. The upper left second bicuspid and first molar are to be extracted on account of disease of the peridental membrane and a bridge is to be placed from the second molar to the first bicuspid. Radiographs of these roots are to be ordered. Both third molars are missing.

In the lower jaw, the septal tissues between the front teeth have been injured by the misuse of the toothpick (71). There are deposits of salivary calculus on the lingual of the six front teeth. Both third molars, the left first molar and the right bicuspids are missing, the remaining molars and left second bicuspid are to be extracted on account of disease of the peridental membrane. Three of these teeth are loose. A partial denture is to be made replacing the bicuspids and molars on the right side and the second bicuspid and two molars on the left side. This plate is to have a lingual bar and clasps on the right cuspid and left second bicuspid.

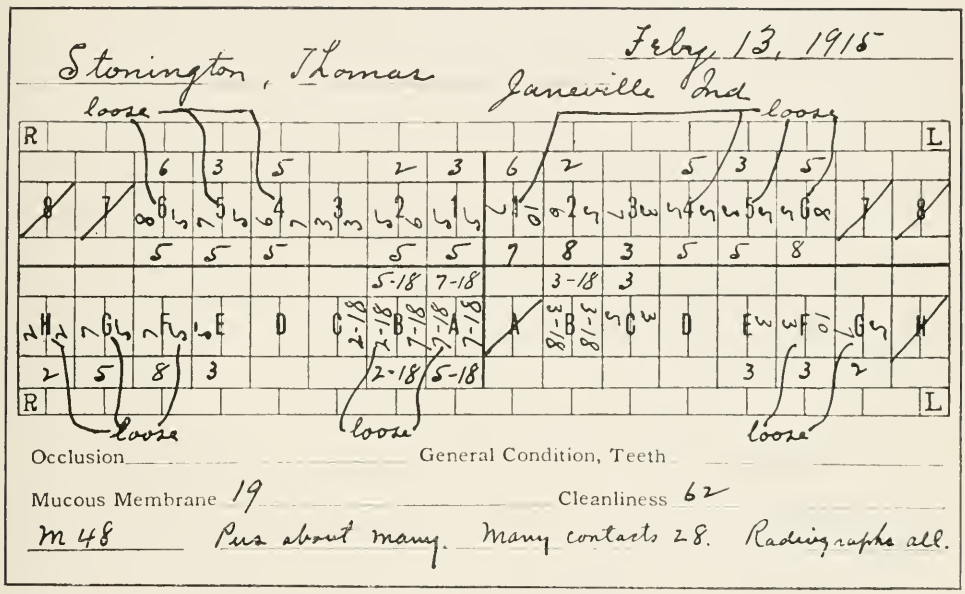

Fig. 506.

Fig. 506. Record of Examination. (Marks from Table No. 2.) The above is a reproduction of the record of the examination of the mouth of a man of forty-eight, previous to the making of the radiographs shown in Figures 234 and 235. The effort was made to record the depth of the pockets on each side of each root, measured from the gingival line, and the figures to the mesial and distal of each tooth may be compared with the absorptions of bone shown in the radiographs. It will be noticed that pockets were not marked about the lower first bicuspids, and the bone about these teeth is normal or very nearly so in the radiographs. This will give a good idea of the value of such a record in showing the actual conditions.

This patient had been practically immune to dental caries all his life, and had never taken proper care of his mouth. Cleaning was unnecessary for caries, and, like many persons who are apparently so fortunate, his habitual lack of care could not be changed to prevent the loss of his teeth later in life from disease of the investing tissues. It will be noted that there were deposits of salivary calculus (18) about the lower incisors, and deposits of serumal calculus (19) generally about the teeth.

As noted in the descriptions of Figures 234 and 235, this patient was advised to have all of his teeth extracted.

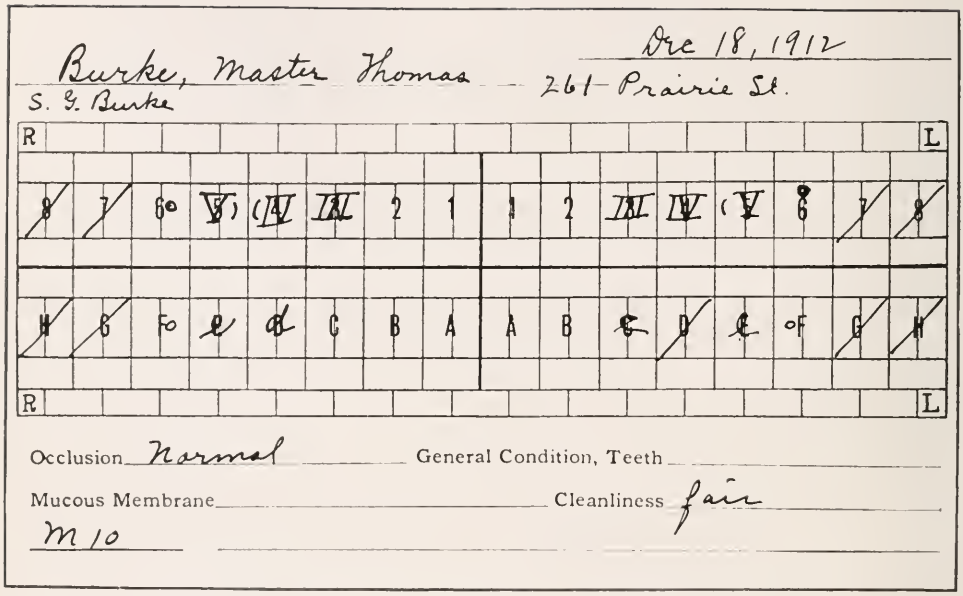

Fig. 507.

Fig. 507. Record of Examination. This boy of ten years was taking fair care of his teeth. His occlusion was normal. All second (permanent) and third molars are unerupted.

In the upper arch the temporary cuspids and first and second molars are in place (indicated by roman numerals). There are three proximal cavities in these, and an occlusal cavity in the right first molar. There is also a buccal pit cavity in the left first molar.

In the lower jaw, on the right side, the temporary molars remain (indicated by script lower-case letters), and the first molar has an occlusal cavity. On the left side, the temporary cuspid and second molar remain; the temporary first molar has been lost and the bicuspid has not yet erupted. There is an occlusal cavity in the permanent first molar.

If such records are kept of the mouths of many children, made for each child every three or four months, they will come to furnish very valuable information, not only of the time of the loss of the temporary teeth and the eruption of the permanent teeth, but of the general progress of cases, the ages at which decays of various classes occur, etc.

greatest importance to determine how much of the end of the root, if any, has been denuded. This is best done with the sharp steel probe, and the extent of the cavity in the bone, as well as the condition of the bone itself, may be made out.

The examination for pulp conditions will usually be in response to some complaint by the patient. These conditions have been discussed. It is only necessary here to call attention to the fact that in cases in which the pulp is alive, the pain may not be definitely located by the patient, unless there is an open cavity to direct the attention to the tooth. On the other hand, such a cavity may lead the patient into error in locating pain. Thermal changes and actual contact of food, or an instrument, are the most certain means of locating the teeth involved. It should be remembered that the differential diagnosis between hyperemic and inflamed pulps is based on actual exposure.

In cases in which the inflammation of the pulp has extended to the periapical tissues, the tooth is easily located by the fact that it is tender to touch, as by tapping it with an instrument. Discolored teeth should call for inquiry as to previous treatment, to determine whether the pulp has been removed, or the tooth contains a dead pulp.

Lastly, one should look to the condition of the hard tissues of the teeth, recording areas of erosion, atrophy or hypoplasia, abrasion, and caries. In this connection missing teeth should be noted, and inquiry made to learn if they have been extracted, or have failed to erupt.

The examination for caries should be separate for three groups of cavities. First, one should look for pit and fissure decays, using an explorer and mouth mirror. In addition to the occlusal surfaces of bicuspids and molars, the lingual surfaces of the upper lateral incisors and the buccal and lingual surfaces of molars should always be examined for decays of this class. Second, the gingival third positions should be examined. These should be dried for this purpose, either with the air syringe, or by laying a roll of gauze on either side of the arch. Many beginning decays, which will not be discovered with an instrument, nor observed if moist, will appear as whitened areas when dry. When discovered at this time, their further progress may possibly be prevented. Third, should come the examination of proximal surfaces. If the teeth are dry, either whitened surfaces or discolorations showing through the occlusal plates may indicate decays. The pair of right and left explorers will

usually locate proximal cavities unless they are very slight. A silk floss should be carried through each contact. It will usually be cut or frayed, or will drag past the contact, if there is a decay. Many proximal decays which can not be reached with instruments, will be located with the floss. If a second attempt with the instrument fails, a separator should be placed to move the teeth apart sufficiently to give room for the explorer.

This will complete the examination. The directions given may seem a little long, but if followed for a time until a routine habit has been acquired, such an examination may be in the average case carried out to the last detail and fully recorded within about ten minutes, if an assistant makes the record. It will require a few minutes more if the dentist must himself record his findings.

The Record of the Examination.

The plan to be followed in recording all of the many little details of a thorough mouth examination must be so simple as to be easily made with very slight expenditure of time. In fact, the assistant should be able to make the record as the examination proceeds. A plan which is too elaborate will be impractical. It has been my effort to develop a scheme fitted to the regular routine examination, with definite means of recording cavities of decay, the condition of the investing tissues and of the pulp, as they commonly present in practice, by the use of certain symbols, figures and letters. Provision is also made for the entry of other data which are frequently desirable or necessary. Two tables of symbols, figures and letters are given; one of these provides for only partial detail in entering causes of inflammations of the gingivæ, the other for complete detail in recording these. These tables are so arranged that one may use the more simple one for a time and then change to the other, or parts of both may be used without confliction or confusion.

The record of the examination should include all of the conditions found, with whatever of the previous history may be necessary to a complete diagnosis; the treatment determined upon; and a memorandum of the fees to be charged, if that question is discussed. Every item which might be of value for future reference is thus entered at the time. The subsequent use to be made of this record will be explained.

This plan has been gradually developed during the past fifteen years and has been used more or less fully by a goodly number of practitioners. While it is by no means as perfect as might be desired, it will enable one, with a little experience, to make a very exact record without loss of time. The plan is referred to in papers written in 1904* and 1912.†

THE EXAMINATION CARD.

The record is made on a 3 x 5 inch white card. (See Figure 501.) As will be noted by reading the description of the card, the upper teeth are represented by figures, the lower teeth by letters. There is a distinct advantage in this, as compared with diagrams of the various teeth, as it permits the making and transferring of the record of a single tooth, so that entries may be made in a daybook and transferred to a ledger or record card or book. It will be noticed that there is a rectangular space for the purpose of recording separately the condition of the soft tissue on the mesial, buccal, distal and lingual sides of each tooth. There are two rows of rectangular spaces, one for the upper jaw and one for the lower, in which to record the condition of the contact which is responsible for inflammation of the interproximal gum septum — the septal tissue.

Aside from the general conditions which may be indicated on the lines below the diagram, and on the reverse side of the card, three sets of markings may be made. Numbers are used to indicate the condition of the investing tissues, letters to indicate the condition of pulps, and symbols for decays and operative procedures. These provide a definite plan of recording practically all conditions ordinarily met with in dental practice. The numbers indicating the inflammations of the investing tissues are particularly important, as the area may not only be definitely located but the cause may also be indicated. If the operator has before him a chart containing these numbers he will very soon become familiar with most of them. The use of the various numbers, letters and symbols is fully explained in the descriptions of the accompanying illustrations. (See Figures 502 to 507.)

THE USE OF THE CARD. When a patient presents for examination, the name, address, and possibly the name of the person

* Keeping of Dental Records and Accounts. Northwestern Dental Journal, Vol. 2, p. 15.

† Preventive Treatment of Peridental Disease, Dental Review, Vol. 26, 1912, p. 801.

who referred the patient, are entered. Then as the examination proceeds, the dentist, with the chart before him as a guide until he becomes familiar with it, may mention the conditions found, while the assistant records them. For example, he may direct her to enter under "General Conditions" the number 62 (see Table No. 2) which would indicate that the patient's care of the mouth was generally poor. It is not necessary that the patient know the meaning of the number. Or, if there should be a general recession of the gingivæ, without apparent cause, the number 17 would be entered under "General Conditions." During the more critical examination of each jaw, deposits of salivary calculus will frequently be found on the lingual surfaces of the six lower front teeth, and the assistant would be asked to mark the figure 18 in the proper places; or if deposits are present on the majority of the teeth, the figure 18 may be entered under general conditions. If there is an inflamed septal tissue, the number indicating both this fact and the cause should be entered in the proper place. Detachments of the peridental membrane may be definitely recorded, with the depth of the pockets. Deposits of serumal calculus should be noted. In this way every item in the examination is recorded. Occasionally something will be observed that can not be recorded by any of the numbers, letters or symbols in the tables, and it will be necessary to make a brief written memorandum on the card.

It is desirable to enter the patient's age. Some patients are inclined to resent being asked their age by the dentist, and as it is not important that the exact age be recorded, I generally guess at the age for adults, and make the entry as M36 or F23, indicating the sex and age. This is entered in the lower left corner, although the word *age* does not appear on the card. Patients will often look over these cards and they will not understand the entry, so there will be no criticism if the dentist has guessed too high.

Fillings and other operations to be performed are entered as indicated in the illustrations. If there is a discussion of fees, a record should be made, on the reverse side of the card, if necessary, which will specify the operations covered by the fee mentioned, so that there will be no misunderstanding later on.

The card should be placed in an alphabetical file and on the occasion of each subsequent appointment, it should be laid out on the operating tray, so that the dentist may see at a glance what is to be done. The card will be an aid in recording the

operations performed, and if each is checked on the examination card as it is entered on the patient's record, the card will, on each return of the patient, show the operations which have been completed, and those which are yet to be performed.

When the series of operations is completed, the card is again placed in the file for future reference. On the occasion of the next examination of the same patient, six months or a year later, a review of the case should be made. Locations in which the gingivæ were inflamed at the time of the previous examination should receive particular attention. If there were deposits of serumal calculus about certain teeth, it will be important to know if new deposits have occurred. If certain positions were marked for deposits of salivary calculus or for lack of cleanliness, it will be interesting to note whether or not the patient's care has improved. If so, a statement to that effect should be made; if not, new directions and additional warnings should be given in the effort to secure improvement in the future. If contacts have been built out, or other operations performed to cure areas of gingivitis, the subsequent examination will demonstrate whether or not these were successful. In this connection it is important that the original memorandum shows whether or not there was at that time a pocket, because it will be found that contacts will not remain tight if there has been much detachment of the transseptal fibers, the chief function of which is to hold the teeth in close contact.

Such a systematic plan of handling cases and checking them can not fail to be effective in improving the service of the dentist and in bringing patients to a better appreciation of the value of this service, to the end that they will themselves become more earnest in the care of their mouths.

APPENDIX.

A MACHINE FOR GRINDING MICROSCOPIC SPECIMENS.

ILLUSTRATIONS: FIGURES 508-518.

IN connection with my studies of the pathology of the hard tissues of the teeth and of other hard substances, such as deposits of calculus, bone, etc., I found it necessary to have a machine with which an almost unlimited quantity of hard material could be ground for microscopic study, with the greatest possible degree of accuracy. The old method of cutting a section from a tooth and grinding it down by hand to microscopic thinness was out of all question, as I required hundreds of such sections. A machine for this purpose was not available; I therefore designed one and had it built to order. This at once enabled me to have prepared without delay and with little effort on the part of my laboratory assistant, the finest grindings of the hard tissues, ready for microscopic examination. Without such a machine it would have been impossible to have prepared such sections as are shown in a number of the illustrations. For example, Figures 163 and 259 show beautiful photomicrographs of sections of roots, one with a deposit of salivary calculus, the other with a deposit of serumal calculus attached. These sections were ground to one-half of one thousand of an inch, by placing a thick slice of the material in the machine and adjusting the micrometer gauge for the thinness desired, and turning on the electric current. The machine stopped automatically when the section was ground according to the adjustment of the gauge.

The machine has two mechanisms, one for slicing the material so that the sections may be mounted on a disk, the other for grinding sections so mounted to microscopic thinness. It is with the belief that quite a few members of the profession will be interested that I present a description of the machine, with a number of illustrations, in this book.

The basis of this machine is the larger watchmaker's lathe, known as No. 2. It must swing 4 inches, the length of the bed

must be 12 inches, and be absolutely solid. A test should be made of the alignment of the lathe head to see that this is exact. If there is any inaccuracy, another lathe should be selected. The power should consist of one-fourth horse-power motor, of the type made for the dental laboratory bench. This power should be transmitted to the lathe through an overhead shaft of a length that will give good room to operate the lathe without the motor being in the way, as shown in Figures 510 and 511.

THE SLICING MECHANISM.

This is an arrangement for slicing very hard substances which can not be cut with the ordinary steel saw — such as the enamel of teeth, silicified fossils, rocks, etc. (See Figures 508 and 509.) This consists of an aluminum disk fitted to the lathe head, and surrounded by a special form of spatter guard that admits of the use of the periphery for cutting, and an object-holder fixed upon the slide rest of the lathe. The object-holder consists of a clamp that grasps a brass tube slotted at the free end, in which teeth or other objects may be made fast with plaster of Paris or sealing-wax for slicing. Or in place of this a brass mandrel, upon the end of which there is a threaded nipple by which any of the grinding disks may be attached. These are fixed in the position of the ordinary tool post, and may be swung horizontally to any possible position in relation to the aluminum disk. An object can therefore be so placed on the disk as to be cut in any direction desired. Usually these are fixed upon the disk with sealing-wax. In using the aluminum disk it is fed with carborundum powder suspended in soapy water to give it some stickiness. This is applied with a brush by hand, and is kept going so constantly as to prevent the disk from running dry. The ordinary aluminum plate, of twenty-four to thirty gauge, may be used for making these. They are first cut in circles by hand, as large as the lathe will swing (4 inches), and then are cut down to 3½ inches with a tool in the slide rest. These are quickly made when wanted. They wear out rapidly, and yet one of them will do much cutting of very hard substances, and do it accurately and delicately. Rings may readily be cut from the ordinary glass test-tubes without special danger of breaking. The crown of a molar tooth may be cut into many slices; fossil teeth, silicified fossil woods, stones, etc., may readily be sliced as thin as they can be handled in the after-work of preparation.

THE GRINDING APPARATUS.

The grinding apparatus, shown in Figures 512 and 513, is built upon a base fitted to the lathe bed in the same way as the lathe head, or tailpiece. It has one main shaft parallel with the lathe bed, in good and sufficient bearings to maintain accuracy of alignment and perfect steadiness for long continued usage. This shaft moves freely lengthwise, or backward and forward, while turning slowly in its bearings. On the end of this shaft next to the lathe head — the forward end — there is a larger portion, or ring, and this end terminates in a threaded nipple, upon which the removable grinding disks are screwed firmly against the face of this larger ring, to secure accuracy of adjustment. The use of these disks will be more fully explained later.

The micrometer adjustment is to the right of the housing for the shaft. (See Figure 513.) A piece is embedded on the lathe bed, but left free to slide back and forth in the length of the shaft; this reaches nearly to the housing of the principal shaft, when it is pushed through as far as it will go. The shaft has a bearing in the front end and back end of this cylinder, upon which the part moves back and forth with the shaft. The shaft is connected by a worm gear to a pulley wheel back of the shaft, which is actuated by a belt on the middle pulley on the overhead shaft. (See Figures 511 and 514.) This allows the piece to move backward or forward at will through its bearings and housings, which are attached to the lathe bed by the thumbscrew below. The micrometer is attached to the front end of the cylinder, which is made solid with the housing of the wheel which turns the shaft. On the front end of this cylinder a nut is placed upon screw threads which forms the micrometer. This nut does not turn with the shaft, and the scale is always before the eyes of the operator. This nut, with the graduations cut in it, is to the left of the worm gear near the right end of the shaft in Figure 513. The movable nut has forty threads to the inch. Moving the nut through one revolution gives twenty-five one-thousandths of an inch, and this gradation is placed upon it. The gradation of the disk is on the same principle as that on the screw calipers used by machinists for fine measurements — one-thousandth of an inch — but as this disk is 1⅛ inches in diameter, the graduations of thousandths are so wide that one-quarter of one-thousandth may readily be used. It differs in plan, in that both the graduation and the parallel lines are placed upon this disk. On the machinist's micrometer the lines are placed on the shaft

and the graduations on the nut. The graduation is read from the side of the finger on the movable nut, and the lines are read from its end.

Turning the screw of the micrometer backward allows the shaft to move farther forward toward the grinding-stone. Moving it forward draws the shaft back from the grinding-stone. The measurement of this movement constitutes the basis of the action of the micrometer. In action the front part of the micrometer engages with a lever from a bar gliding in the automatic cut-off, and this automatic cut-off throws a switch which turns off the current of electricity at a certain point and stops the movement of the machine. (See Figures 515 and 516.) The adjustment of the micrometer is so made that this cut-off will be thrown and stop the machine at the measurement determined upon for the finishing of the grinding, and for which the micrometer is set. When this switch is thrown by the forward movement of the shaft the motor and the whole apparatus stops. At each time the switch is thrown it must be reset to turn on the current and start the machine again. It is a very perfect micrometer. It may be seen in Figures 512, 513 and 514 at the right end of the shaft. The adjustment will be given in detail later.

The forward movement of the shaft when grinding, and also the pressure exerted upon the stone, are furnished by a tailpiece placed behind it and attached to the lathe bed. (See Figure 512.) This has a plunger actuated by a spiral spring, which pushes the shaft forward toward the stone. The amount of pressure exerted in the grinding is controlled by the amount of compression of this spring in fixing the piece to the lathe bed. It may be much or little, as desired. Usually very little pressure is required. The further arrangement for finding this measurement will be described later.

On the rear portion of the graduated disk, or wheel, a portion or space is toothed, and connected with a worm pinion or threaded shaft by which the main shaft is turned in its bearings. A belt is attached over a wheel on the end of this worm shaft, and extends to the middle wheel, previously mentioned, on the overhead shaft. When this belt is adjusted and the motor started, it causes the main shaft in the grinding machine proper to turn slowly on its axis, while being pressed against the stone by the tailpiece. By this arrangement every part of the specimen fixed on the grinding disk is brought successively against every part of the rapidly revolving stone, and is cut perfectly level in all of its parts. (See Figures 512 and 513.)

The grinding disks. The grinding disks are of brass, accurately turned ⅜ inch thick, and 1⅜ inches in diameter. They have a threaded hole ¼ inch deep in the back to fix them to the nipple on the forward end of the shaft of the grinding machine. A machine should have a half-dozen or more of these, lettered or numbered on the edge, so that records of each may be made when measuring preparatory to mounting specimens for grinding. As the mounting of specimens on others of these may proceed while the grinding on one is going on (for the machine, being automatic, needs little attention), at least six are necessary for rapid work.

The machine may be stopped and the disk removed from the shaft by a few backward turns, the progress of the grinding examined, the disk returned for further grinding, etc., at any time during the progress of the work. The face of the disk, which should be perfectly flat and parallel with the face of the stone, should always be perfectly bright, so as to reflect light through the specimen when it becomes thin. This enables one to judge very closely of the thickness by the eye (after sufficient practice), which sometimes proves a valuable check on the setting of the measurement in the beginning.

The point finder. This is a piece of steel one-eighth of an inch thick, fitted to the lathe bed and set against the face of the lathe head, and made fast by a thumbscrew passing through the lathe bed from below. (See Figure 513.) It has a strong arm which passes around other fixtures between the lathe head and the forward end of the base of the grinding machine. It is provided with a set-screw, by which a range of variation can be made in the distance of the forward end of the frame of the grinding machine from the lathe head. When this is in place and the measurement of a disk has been made and recorded for the grinding of a specimen to a specified thickness, the machine may be taken to pieces and set up again and the grinding proceed without fear of disturbing the measurement, so long as the set-screw in the point finder is not moved. It is often necessary during grinding to loosen the grinding machine from the lathe bed, slide it back to adjust something, to remove disks for examination of the progress of the work, etc. This point finder, by preserving the distance between the lathe head and the grinding machine, enables one to do this at will, and again find his exact point of measurement simply by sliding the frame of the grinding machine forward against the set-screw of the point finder.

This little device seems absolutely necessary to the highest usefulness of the machine.

Lap wheels. I began my work of grinding specimens by the use of lap wheels, but soon discarded them because they were dirty. They cut much faster than stones, however, and may be used for the bulk of the work when much grinding of very hard material is to be done. They are not necessary in grinding teeth, bone, etc., but in grinding the harder fossils, especially those impregnated with the silicates, and in some geological work, they become necessary.

The best lap wheel I have used is an aluminum wheel. Brass or iron will do the work, but aluminum holds the grit better, cuts with lighter pressure, and does the work more quickly. In using these I have fed them continuously by hand with carborundum powder in soapy water, using a brush.

Grinding-stones. Any one who is doing much grinding should have a good supply of stones. I have a pair of carborundum wheels, a pair of emery wheels, a pair of India oil-stones, and a pair of Arkansas stones. In each of these pairs one is fine and the other coarser grit. Every stone is dressed to a perfect face on the lathe head where it is to do its work, with a black diamond held in the slide rest.

These stones, when put in good shape, seem capable of doing an unlimited amount of work. The conditions of the grinding prevent them from getting out of true. All that seems necessary is to roughen them a bit with a picking wheel when they become too smooth to cut well. For this purpose a much smaller picking tool than the smallest sold for the general mechanical uses seems desirable. This picking wheel has sharp teeth of the hardest steel possible on its periphery. It is held in a handle in such form that the wheel is free to turn. In use it is held against the rapidly rotating stone and slowly passed over its entire surface. It may be held in the hand, aided by a tool rest, or may be arranged for use in the slide rest, which is the better form for this work.

Watering the stones. In grinding, the stones are kept wet in running ice-water. A balsam that is too soft to hold a specimen for grinding in water at room temperature will hold it perfectly in ice-water, because it is much harder when cold. For this purpose, a bucket or a large rubber bag is hung on the frame which holds the overhead shaft, and is filled with bits of ice and then filled with water. Both the ice and the water must be clean, for the opening in the tube where it passes the valve which regu-

lates the flow is very small, and a small particle of dirt might stop the flow. In this case the specimen being ground would be burned instantly. An ordinary rubber tube conducts the water, and is connected with a metal tube having a brush attached to the other end. This tube is mounted on a stand and the brush may be placed in any desired position to deliver the water to the stone. This metallic tube is provided with a valve for the regulation of the flow. The brush is made upon a short tube fitted into the end of the metal tube. To make this brush, the plain part of the small brass tube is first covered with thick shellac dissolved in absolute alcohol. A layer of the bristles are then placed around it and wrapped tightly with a fine, strong thread. More shellac is applied and another layer of bristles added. This is continued until the brush is large enough. Then it is wrapped thoroughly with a cord in shellac, allowed to dry, and then trimmed. Two of these have served for six years of fairly hard usage.

Waste water. A spatter guard is made by bending a five-eighth-inch round brass tube into a circle, the inner diameter of which is the size of the stones used, and brazing the ends solidly together. This is fixed in the lathe and one-fourth of its inner circular diameter is turned away. The grinding-stones will go inside this. This piece is provided with a foot and hollow post and fitted to the lathe bed with a washer and nut, the same as other pieces are attached. This catches all waste water and through a rubber tube attached to the end of its hollow post under the lathe bed delivers it into a convenient receptacle. This prevents all of the spattering of water which would otherwise be thrown from a rapidly revolving wheel. If it should be inclined to run over when a very full stream is wanted, a piece of rubber dam may be stretched over the foot and pulled to its upper end. This may be caught under the guard in fastening it to the lathe bed, and will deliver any overflow into a receptacle placed to receive it. In this way nothing is wet or spattered with water.

Preparation of material.

In the preparation of material, such as teeth, bone, etc., in histological work of ordinary delicacy, the specimen is first ground flat on one side by hand on a rough stone 4 inches in diameter, on the motor, and finished perfectly flat on one of the finer stones on the lathe head. The piece is then washed clean and placed in absolute alcohol for a sufficient time to remove all traces of water, or, when cracking or injury from shrinkage is

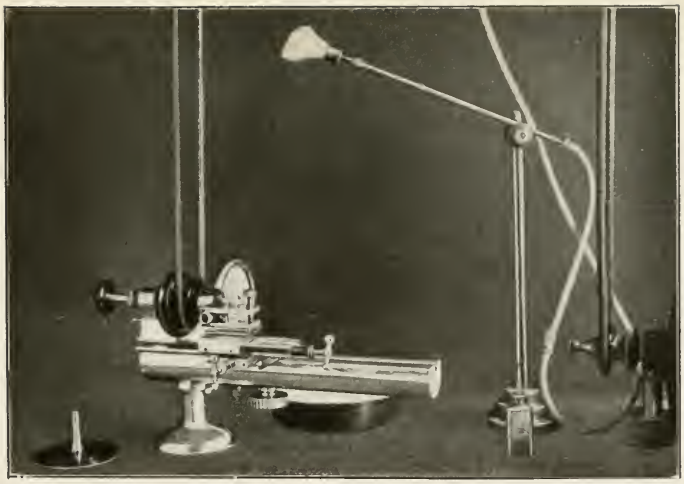

FIG. 508.

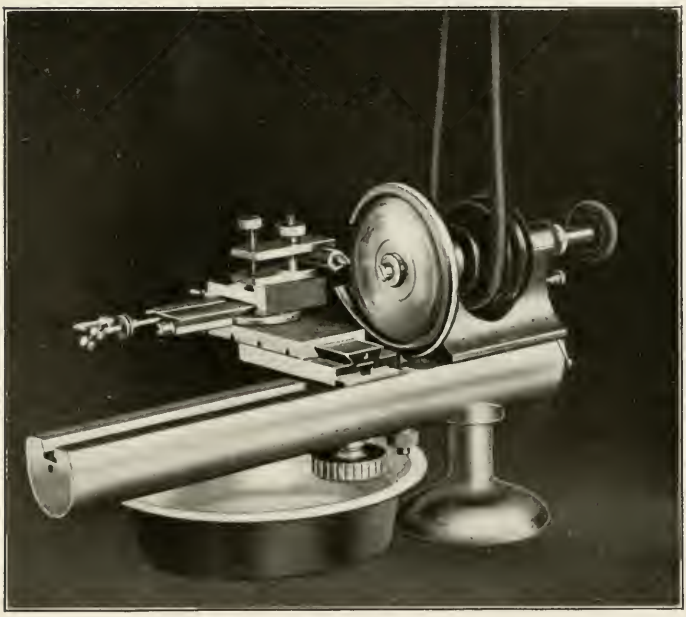

FIG. 509.

FIGS. 508 AND 509. Lathe with mechanism for slicing very hard material. Figure 508 is the more ordinary view of the machine with the slide rest and object holder in position. In Figure 509 the lathe is turned about to give a better view of the slide rest, object holder, spatter guard, and aluminum disk. A general view, showing the arrangement for transmission of power from the motor is shown in Figure 511. In Figure 509 the slotted tube used to hold the object being cut may be seen close to the cutting disk. Water from a rubber bag hung above (See Figure 511) is conducted through a rubber tubing to the adjustable metal tube on which the brush is mounted. The disk used for cutting is surrounded by a spatter guard which is open for a space at one side so that the periphery of the disk may be used in cutting. This guard gathers all water and grit, and delivers it into a pan below through a hollow post to which a tube is connected. When doing this kind of work all of the bearings of the machine should be carefully swaddled (wrapped) to keep them safe from intrusion of grit.

Fig. 510.

Fig. 510. General view of the grinding machine in operation. It is shown better in Figure 511 and is there described.

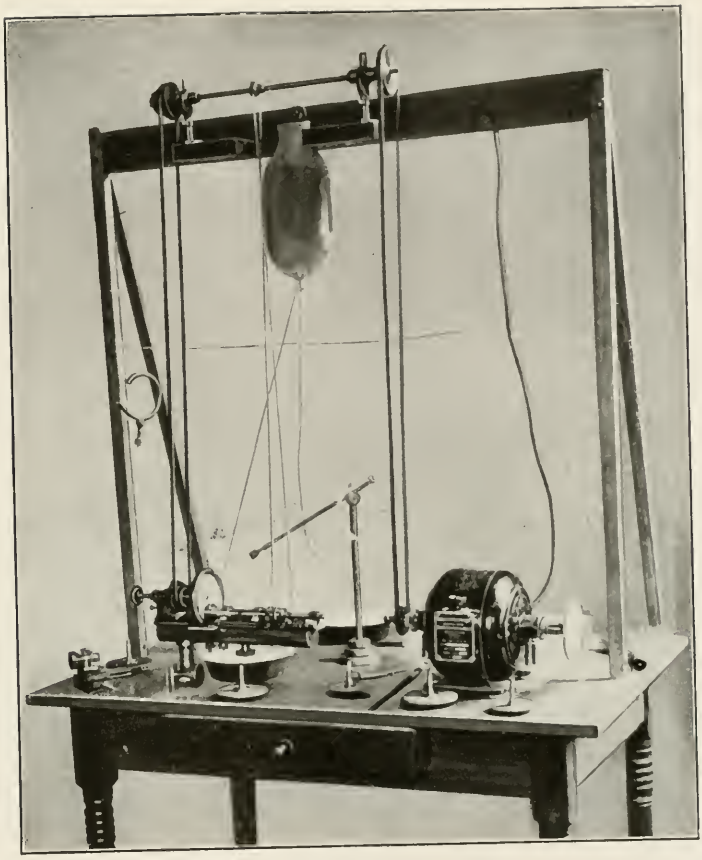

FIG. 511.

FIG. 511. A general view of the grinding machine, showing particularly the arrangement for transmitting the power from the electric motor to the lathe. The lathe to the left is a watchmaker's lathe No. 2. The bed is 12¼ inches long, and it swings 4 inches. The power is furnished by a ¼ horse-power motor, transmitted to the lathe through an overhead shaft. The pulley on the left end of the motor shaft is brass, 2½ inches in diameter, and carries a ¼ inch belt. The pulley on the right end of the overhead shaft is 5 inches in diameter, thus reducing the speed one-half and doubling the power. On the left end of the overhead shaft is a pulley which is a copy — reversed — of the pulley on the lathe head, which has four grooves. This gives good varieties of speed with each speed of the motor. The third pulley near the center of the overhead shaft is for the purpose of rotating the disk carrying the specimens while they are being ground.

Water is delivered to the grinding stone from a rubber bag or bucket hung on the frame above, through a rubber tube to the metal tube on a movable stand, which may be so placed as to bring the brush at its end against the stone. This stand and brush are better seen in Figure 512. The water is conveyed from the lathe by another tube (not shown in this illustration) which may be seen in Figure 512.

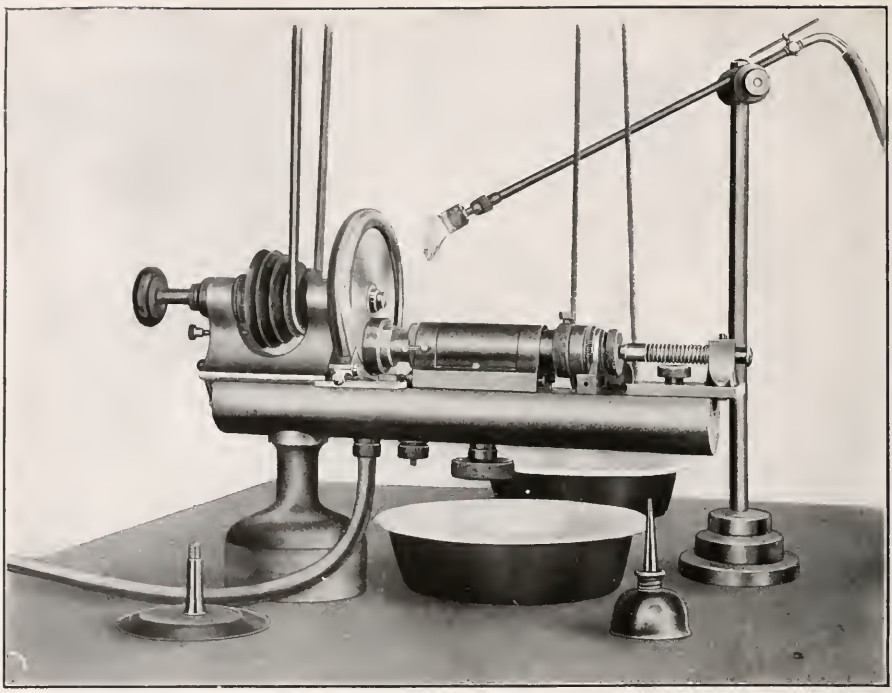

FIG. 512.

FIGS. 512 AND 513. The lathe with the grinding machine mounted upon it in position for work. On the left next to the lathe head is the grinding stone surrounded by the spatter guard, which gathers all of the water from the wheel and delivers it through its hollow post into a rubber tube below the lathe bed, which conveys it to a conveniently placed receptacle. The water comes from a rubber bag or bucket hung on the overhead frame (See Figure 511) through a rubber tube to the metal tube mounted on a movable stand so that the brush through which it passes may be placed against the stone. The grinding machine proper is secured to the lathe bed by the larger thumb-screw seen below. The point-finder is seen at the foot of the spatter guard, and is secured by the middle thumb-screw seen below the lathe bed.

The shaft of the grinding machine (6 inches long) runs through its whole length, but is completely covered in by its housings to protect its bearings from grit, except at its forward end (next to the grinding stone). This part is protected by a swaddle held by a ring, which keeps the working bearing clean. On this end the grinding disk is seen almost touching the stone. The micrometer surrounds the other end of the shaft, but does not touch it. It is connected with the back end of the machine, which is freely movable backward and forward, carrying the shaft with it. In the back part of this is a toothed wheel made fast to the shaft. This is actuated by a worm drive on the shaft of a pulley wheel driven by the middle one of the belts descending from the overhead shaft in Figure 511, the right-hand belt in Figure 512. This belt passes over a wheel, which may be seen in Figures 514, 515 and 516. Pressure for the grinding is supplied by a plunger actuated by a spiral spring seen at the extreme right hand in Figure 512.

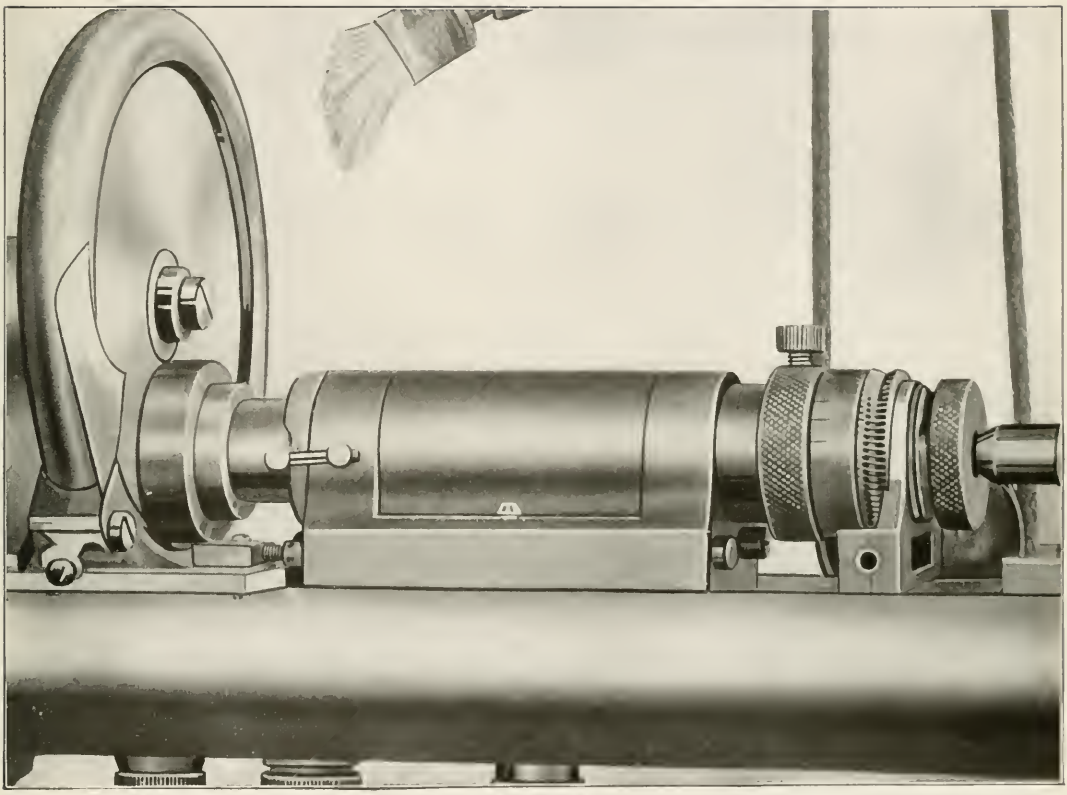

Fig. 513.

Fig. 513. The grinding stone and shaft of the grinding machine.
For description, see opposite page.

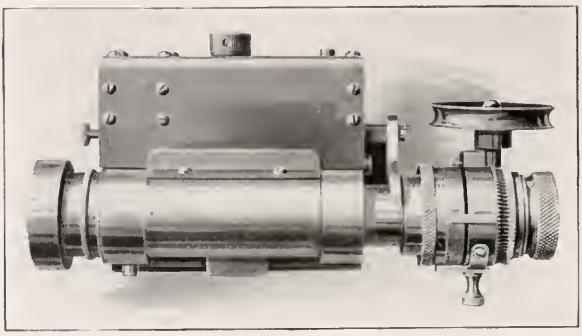

FIG. 514.

FIG. 514. View from above of grinding apparatus with the electric cut-off. The housing for the electric cut-off is above the shaft in the illustration. The button above this housing (on the back side of the machine) is set with the fingers to start the machine after it has automatically broken the current and stopped the machine. The pulley wheel to the right of the illustration is connected with the middle pulley on the overhead shaft. This pulley shaft engages the worm drive on the grinding shaft, causing it to revolve. To the left end of this shaft is the grinding disk on which the specimens to be ground are attached. The action of this in relation to the stone is explained in the text. The mechanism of the electric cut-off is shown in Figures 515 and 516.

The return current from the motor is diverted to pass through this cut-off by plugging into the two large holes to the left side of the mechanism. The respective poles are connected through the mechanism with the two plates which are situated behind the two bars, the ends of which overlap near the center of the mechanism in Figure 515. The plate back of the left or fixed bar is seen better in Figure 516. In this illustration, the other plate has snapped down with the other bar. These two bars are of hardened steel, with accurately ground chisel edges. There can be no arc of the electric current between these bar ends, as the contact of the plates, which are wider, is not broken until after the bars have separated.

As the shaft of the grinding apparatus is moved toward the stone by the spring of the tailpiece, one of the steel bars moves with the shaft, and when the movement has continued to the point determined by the adjustment of the micrometer, the chisel edge of the movable bar will pass the edge of the other bar, and a spring causes the bar to the right in the illustrations to snap down, carrying the contact plate with it, thus breaking the current and stopping both the grinding stone and the shaft. The spiral spring shows best in Figure 515. The switch may be reset by the knob on the back of the mechanism.

Just to the left of this spiral spring and in line with it is a flat spring attached at its left end, which bears against a plate in under the spiral spring. This delivers the current from the lower socket to the sliding part, maintaining a pressure contact. The large screw below in both illustrations is for the purpose of attaching the shaft to the lathe bed.

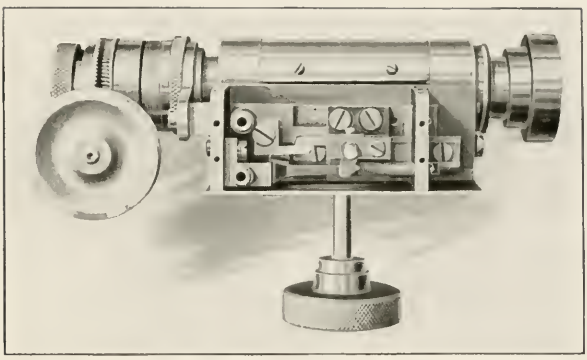

Fig. 515.

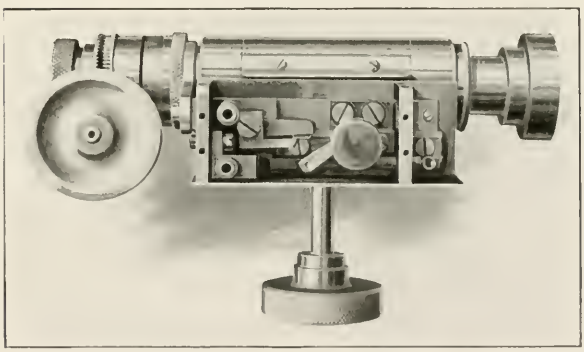

Fig. 516.

Figs. 515 and 516. Views from the back of the grinding apparatus, detached from the lathe, and with the housing removed to show the elastic automatic cut-off. Figure 515 shows the bars set so that the current is on; Figure 516 shows one bar dropped, the current being disconnected. The adjustment of the micrometer is so made that this cut-off will be thrown and stop the machine at the measurement determined upon for finishing the grinding. See description under Figure 514.

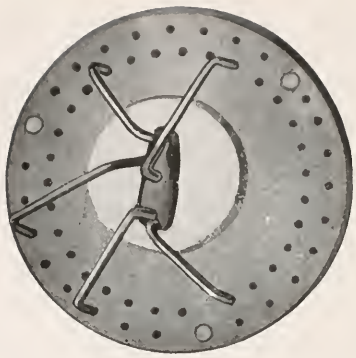

FIG. 517.

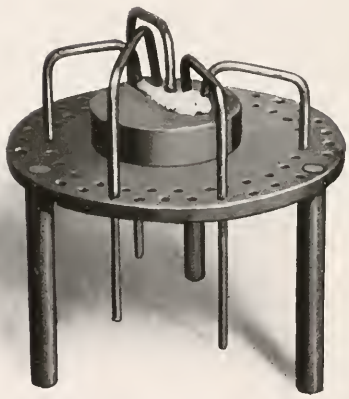

FIG. 518.

FIGS. 517 AND 518. The spider with a grinding disk upon it and a specimen laid and secured by bent rods called "dogs." When these dogs are placed and pressed down through the holes in the disk of the spider, they hold fast. With a little pressure of the finger outward on the end of the rod below the disk of the spider, the dog slips up and is loose. The disk of the spider is three inches in diameter.

not feared, it may be dried in the warming box. Then when dried and warmed to about 120° F., it is ready to mount with balsam on the grinding disk.

Management of balsam. I suppose the management of balsam will always be a difficult problem with many persons. Many, however, learn it quickly. One may take the dry balsam and dissolve it in xylol, and filter it at a high temperature, say 110° F. or 120° F. Or one may use the prepared balsam for microscopic mountings. In either case it must be evaporated until stiff enough so that it will move rather sluggishly at 110° F., but will be fluid at 120° F. or 130° F.

Spiders and dogs. Another bit of apparatus is necessary. A circular piece of steel made flat on the upper surface is mounted on three legs 1½ to 2 inches high. The steel disk should have two rows of holes around its periphery, the one row ⅜ inch inside the other. A hard rolled tool, steel wire, or rod 3/32 inch in diameter, should exactly fit these holes. These rods should now be bent at right angles with a short nib on the end, bent again at right angles, so that the nib will point downward when the free end of the rod is set into one of the holes. The length between these two angles should vary from ¾ to 1½ inches in three dozen or more pieces which should be prepared. The end which goes in the holes should be cut so that it will not quite reach the surface of the table when dropped into the holes with the end of the nib on the surface of the circular plate. These rods are called "dogs." (See Figures 517 and 518.)

With this arrangement a warming box having a thermostat to maintain an even temperature, sufficiently high to soften the stiff balsam, is used. The specimen, the balsam, the grinding disk, and the "spider" are placed inside, and allowed to rest until they have reached the temperature desired. Then, working quickly, a sufficient amount of balsam is placed on the grinding disk, and the specimen laid on it. This should be pressed down until it is seen that all space under it is filled with balsam, but little excess should be used. It is well if this rest so in the warming box for ten or fifteen minutes for the balsam to soak well into the specimen. Then the grinding disk, with the specimens, should be laid on the spider and one of the dogs dropped into one of the holes in the steel plate, which will bring its nib on to a part of the specimen chosen. Then another, and still another, should be placed, each with its nib on a different part of the specimen, so that every part of it may be pressed flat on the disk. Each in turn is then pressed down a little, until all

are exerting about the full force of the spring of the rods without permanently bending them. In this condition the specimen is again placed in the warming box.

Any number of specimens of teeth or bits of teeth, bone, etc., which the face of the disk will hold may be placed on the disk, and all may be ground together. Four to six lengthwise sections of incisor or cuspid teeth may be placed at once, or eight to twelve cross sections. It seems to be best, however, not to load the disk too heavily, and to place nothing on the central part of the disk. In other words, specimens should be placed as near the periphery of the disk as possible, so that each will sweep over the entire breadth of the stone. Four lengthwise sections will be ground better than six, as a rule.

After the loaded disk has remained in the warming box until all balsam that will come has been squeezed out from under the specimens, the excess of balsam should be very carefully removed close up against the specimens. The best instrument for removing this is a wooden toothpick with a flat end cut squarely across. When this is pushed against the balsam it will rise up on the toothpick, and it may be wiped away with a cloth held in the other hand. Nothing clogs a stone and stops its cutting more effectually than balsam smeared over it, and all excess which may come in contact with the stone should be removed.

The spider should then be returned to the warming box for from one to four hours, so that it may dry a little about the margins. Then it may be removed and allowed to cool, and await convenience in grinding. It should, however, remain secured on the spider by the dogs if it is to wait more than a few hours, for the disposition of dentin to warp in drying may pull some part of the specimen from the disk. Under these conditions, two or three days, or a week, will do no harm.

When the grinding is completed, the disk is removed from the machine and the specimens flushed with clean water, and dried by the pressure of a soft napkin folded to several thicknesses, or clean pieces of waste-cotton fabric may be used. Then the disk, with its specimens, should be laid in a dish, and sufficient xylol added to cover it, and allowed to rest until the balsam has been dissolved and the specimens released. This will usually require from twenty to thirty minutes, or sometimes as much as an hour. When the specimens are very thin they loosen much quicker than when thick. Any material not penetrated by xylol, as silicified petrifactions and stones, require much more time.

When the specimens have loosened, they are ready for permanent mounting for microscopic study.

Rapidity of grinding.

In order to make rapid progress in grinding specimens, one should have six to ten grinding disks, nearly as many spiders, and a large supply of dogs. The machine is so nearly automatic in its action that it needs but little watching, so that the preparation may be going on while the grinding is in progress. One of the principal points which needs attention is the flow of water. But if the water and ice placed in the receptacle are clean and free from dirt which may stop the flow of water, the only care is that the quantity of water is kept up. The vessel should be large enough to hold a supply for several hours. If the stone should run dry, the specimen would be destroyed in a few seconds.

Setting the measurement of grinding disks.

When beginning a series of grindings, the first thing of importance is to try out and obtain a record of the measurements of each grinding disk for the particular stone that may be selected for finishing. I find that most persons, after some practice, prefer to use a fine stone for the entire grind. In grinding teeth, after roughing down the surface which is to form the specimen, the back is also ground away to a flat surface, which will better accommodate the placing of dogs in mounting on the grinding disks. These may be made quite thin and reduce the grinding with the fine stone. The stone selected is placed in the lathe head, seeing to it carefully that the face of the stone is clean. The grinding machine is brought up in contact with the set-screw of the point finder. Then, with the large nut the shaft is so adjusted that the grinding disk being tried comes close to the stone but does not touch it. The machine should be started and its running carefully noted. While doing so the adjusting nut of the micrometer should be moved one-thousandth of an inch at a time backward to lengthen the shaft, listening for the first touch of the disk to the stone. The moment this is heard, if the machine does not stop, it is because the adjustment is such that the stopping device is not thrown. In that case the nut of the micrometer is screwed forward and tried again until the machine is stopped exactly when the first scrape upon the stone is heard. The click of the switch which stops the current will also be heard at the same time. If, on the other hand, the click

of the switch is heard before the disk reaches the stone, the micrometer nut must be screwed backward to increase the length of the shaft just enough so that the click of the switch is heard at the same time the disk touches the stone. A little practice will make the adjustment easy.

After this is completed for any given stone, or for all of the stones, this record is used for the setting of disks for grinding. This record is made upon a white card and hung where it will be before the operator. At each placement of a disk it may be tried out before the setting of the specimens upon it, placing the adjusting nut at the recorded measurement to see that it is exactly correct. Then the adjusting nut is turned forward as many thousandths as the desired thickness of the specimen and made fast by tightening the brake. Then the grinding may proceed, first seeing that the ice-water is running, and will not require any special notice until the machine stops. Then the disk is dried off and placed in the bath of xylol to dissolve off the specimen. Another disk is adjusted and the grinding proceeds.

Recently an assistant in my laboratory was grinding sections of teeth, and he made all of the preparations, preparatory grindings, disk mounts, and ground and removed from the disks ready for mounting, forty full-length sections of central incisors in six hours, and had his luncheon in the meantime. Every section was complete, of even thickness in every part, and all practically the same thickness. During the past winter six hundred specimens were ground without changing the micrometer. It was then discovered that the specimens being ground were too thick, and a change of 1/1000 of an inch was found necessary to make them thin enough. This difference represented the wear of the stone in that series of grindings. It will be seen that this is not very much, but it is an item which requires special notice.

Grinding frail material.

While the machine facilitates the production of the more ordinary sections to such a degree as to be indispensable to one having many grindings to do, it is in the production of sections of very frail material that the grinding machine stands out as vastly superior to the other methods of grinding. In the study of caries of enamel in which disintegration has rendered the remaining tissue very frail and likely to fall to pieces before it is sufficiently thin, we may obtain the required thinness and yet retain all of the tissue. I have also produced exceedingly fine sections of salivary calculus, and equally good sections from

small crumbs of serumal calculus. The production of these is slow, but fairly certain of good results.

Also in grinding sections of fossil teeth, fossil woods, and the like, in which very fine sections are too brittle to be handled in any way except as stuck to glass, the machine gives excellent results. In geological work it practically removes the difficulties. Good sections of the very brittle stones can be made with fair safety by grinding on the cover-glass.

Much very desirable material for microscopic investigation is so frail, or at least so brittle, when reduced to sections thin enough for microscopic investigation, that it will crumble to pieces, either in the grinding or in the mounting, by the ordinary procedures. For grinding and mounting such material the following processes have been slowly evolved. These may be divided into the balsam process and the shellac process. Material may be made fast to a cover-glass and ground in hard balsam, if it is not liable to go to pieces when this hard balsam is softened by sticking the specimen and glass cover to a glass slide. If, however, the different parts are liable to separate and change position when the balsam softens, shellac should be used for the grinding. I have been much disappointed by failures in grinding in hard balsam rare specimens of enamel which had no cementing substance between the enamel rods. When the softer balsam was added to mount the specimens on the glass slides, the hard balsam was softened and the enamel rods floated out of position. All such material as will not hold together should be ground in shellac.

The use of balsam. To grind in hard balsam, the one side of the specimen may be ground flat on the rough stone and then dried out in absolute alcohol. The ground side should be saturated to sufficient depth with soft balsam, and laid aside until the balsam has become hard enough to grind smoothly. Then the grinding and polishing of this first side should be completed by grinding away all balsam from the immediate surface, and sufficiently into the substance of the specimen to produce a clean, smooth surface of the material. When this has been done, and the surface dried, it should be mounted on an ordinary cover-glass, the thickness of which should have been measured and recorded. In this mounting the cover-glass should be laid on a spider and weight enough placed upon it to insure a perfect fit of the surface of the glass. This should be subjected to about 120° F. for from one to five or six hours, for the purpose of expressing the last bit of balsam possible from between the specimen and

the cover-glass. Then it may rest, awaiting the convenience of the operator, for several days, but the balsam must not be allowed to become "brittle hard," because in that case it loses toughness. All excess of balsam about the margins of the specimen should be carefully removed to facilitate the hardening of that which remains, and especially so that it may not come in contact with the grinding-stone, stick to its surface, and interfere with the cutting.

Good judgment must be acquired by practice as to the hardening of balsam and shellac in these grinding processes. The best idea of it that can be given in words is this: The balsam or the shellac must have become firm enough so that it will not drag or allow the specimen to move while grinding in iced water. Neither must it become hard enough to become brittle, for then it is liable to break.

When ready, the specimen is mounted on the grinding disk. This is done by first cleansing the disk, finishing with xylol, and then sealing the cover-glass to this with soft balsam. This should be placed on the spider and well weighted down with dogs. All excess of balsam should be carefully removed from the margins of the cover-glass. This may be quickly dried at 120° F., or more slowly at room temperature. It should, however, be warmed for a half hour or more, for the purpose of expressing as much balsam as possible. This cover-glass will be well held for grinding in iced water with only a little drying about the margins, if all excess of balsam is cleaned away closely. The balsam should not become very hard.

If the specimen is of considerable bulk and of a quality of material that can be cut with a steel saw, the disk may be caught in a vise with leather-cushioned jaws to avoid bruising, and the bulk of the material removed with a jeweler's saw, leaving only a moderately thin section for grinding. Or if the material is very hard, as stones, silicified fossils, etc., the disks may be mounted upon the slide rest and cut with the slicing disks, previously described.

The specimen is now ready for the final grinding. The record for measurement with the particular stone to be used in finishing has been made, tried out on unimportant material, and the cover-glass has been measured and its record made. With this data, the disk is screwed to its place, the micrometer turned to the proper measurement for the finish, the iced water arranged, the machine set in motion, and it will do the rest. When coarser stones are used for cutting away much material,

I find those with just a little experience prefer to gauge the amount of the cutting by the eye for the coarse stone.

Removal of the cover-glass from the disk. I remove the cover-glass with the specimen from the grinding disk in two different ways, as seems at the time best.

First, the grinding disk is placed on a heated piece of metal that will warm the grinding disk quickly. A stick of rather soft wood, the end of which is cut to a sharp angle and thinned down almost in the form of a blade, is held ready. When the grinding disk begins to warm, the margin of the cover-glass is caught with the end of the stick and steady pressure is made. As the disk warms, softening the balsam, the cover-glass will begin to move under the steady pressure, slowly at first, but more rapidly later, and will slide off the grinding disk before the specimen is loosened. For this plan the cover-glass should be pretty strong, one and one-half to two thousandths of an inch thick; otherwise there will be great danger of breaking it. It is well in some cases to run just a little xylol around the margins of the cover-glass and partially dissolve the balsam which has become dryest, before the heating. Great care must be taken not to allow the xylol to spread on to the specimen, for it would loosen it very quickly.

The specimen is then turned downward and placed on a tiny drop of balsam on a glass slide, and quickly pressed down close and level. As the new balsam will soften the old, a spring clip should be quickly applied to hold it steady. The parts of the specimen are less likely to move if this is laid on ice for an hour or more.

The use of shellac. In the second plan shellac is used instead of balsam for hardening the specimen and holding its parts together in the first grinding. This part of the work is otherwise done in the same way. The drying of the shellac usually requires more time than the balsam.

The attachment of the cover-glass to the grinding disk is done in the same way as when balsam is used to hold the specimen on the cover-glass. The grinding proceeds similarly in every respect.

The important difference in the two processes is in the removal of the cover-glass from the grinding disk, and mounting the specimen. Xylol dissolves balsam very quickly; but xylol does not dissolve shellac at all. Therefore, instead of pushing the cover-glass off the grinding disk, the disk is laid in xylol and the balsam dissolved out. In this there is no danger of detaching or moving the specimen if it is carefully handled.

When cleaned, it is inverted upon a glass slide on a drop of balsam without fear of movement of parts of the specimen, no matter how frail.

The preparation of shellac. It is difficult to keep shellac in condition for this work. The dry scales should be dissolved in absolute alcohol so as to make a moderately thick varnish. It should then be filtered at a temperature of 110° F. to 120° F., or made thinner and filtered at room temperature. Great care should be exercised to keep the filtrate from exposure to a damp atmosphere, for it absorbs water readily and then will throw down fine crystals, which destroy its value for microscopic purposes.

After being filtered it should be evaporated in a close warming box at about 110° F. to 120° F., to the consistence of syrup. In doing this it is well to divide the supply into two or three grades — a thinner, a medium, and a thicker solution. The thinner solution will be used for saturating frail specimens before cutting; the thicker solutions for attaching specimens to the cover-glass for grinding, and the medium solution for either purpose, as the material may seem to require.

Grinding from crumbled material.

Often important material for investigation can be had only in very small crumbs, or broken pieces, such as serumal calculus, sands, crumbled bits of strange stones, or mixtures of such material as is found in some of the coarser sands. These, on microscopic investigation, may reveal their origin and throw light upon geological questions. In addition to the ordinary microscopic observation, the polariscope may be turned on these, and reveal facts as to their origin and structure. Also many things will be found in botanical work, such as sections of small seeds, and the like, which will give useful information.

Having done a few of these grindings, especially of the very frail dental material, such as serumal calculus, extremely frail fossil teeth, etc., plans of work more or less well adapted have been developed. For instance, I have obtained excellent sections of serumal calculus, which can be had only in small crumbs or flakes. A small collection of these bits are first immersed for a time in absolute alcohol, or until all air has been removed, if they are dry, or if they are freshly gathered, until all water has been removed. Then a cover-glass is prepared by covering its central part with the thicker solution of shellac, and these crumbs are placed in this, in what seems to be the best position for obtain-

ing sections. These are allowed to soak full of the shellac, under a close cover, and then uncovered to dry up. If some of the pieces seem to need it, more shellac is added from time to time, until the embedding is sufficient. This may be dried at room temperature, or in the warming oven at 110° F. to 120° F. Shellac should not be subjected to much higher temperatures for a considerable time, because continued high temperature for many days together seems to injure its strength.

When this is sufficiently hard for smooth grinding, and before it has become too brittle (determining this point requires some experience), the preparation is cemented to the grinding disk with balsam and ground to such a point as seems most favorable for obtaining sections. This point is to be determined by frequent removal of the disk from the machine and examination of the exposed surfaces of the several pieces.

When this part is done, the cover-glass is dissolved off the grinding disk by xylol. Then another cover-glass is attached to the surface with the least possible amount of shellac. This in turn is dried to the right consistence. Then the last cover-glass placed—that is, the one on the side that has been ground—is secured to the grinding disk with balsam. When this has set it is placed on the machine and the first cover-glass is ground away and the section ground to the required thinness. They are again dissolved off the grinding disk, and may be at once mounted in balsam on the microscopic slide.

By somewhat similar methods I have obtained excellent specimens of the fossil teeth of the mastodon, the dentin of which was so frail as to crumble in the fingers. Yet the finished specimen showed all of the tissues as perfectly as if it had been from a fresh tooth.

Difficulties in grinding.

In the grinding of material enveloped in shellac, or in balsam, either of these materials is apt to gum up the stone and stop the cutting, or render the grinding very slow. For the finishing of any piece being ground when this is noticed, the stone may be much improved by drying it and washing with xylol on a brush, or a bit of cloth, while the stone is slowly revolved. When gummed with shellac, the washing is done with absolute alcohol. In case the stone becomes clogged with balsam the best treatment is to place it in a dish with sufficient xylol to cover it and let stand over night, closely covered. The stone will then be in as good condition as it was at first. If the stone has

become smeared with shellac it should be placed in a dish with sufficient absolute alcohol to cover the stone, and allowed to remain over night.

With much grinding of hard substances, the surfaces of the stones become worn so smooth that they do not cut well. Then the picking tool should be run over the surface until it is perceptibly roughened. This will cause the stone to cut briskly for a time, and at first — following such sharpening — the ground surface of the specimen is likely to be full of scratches. In that case a smooth stone should be used for the finishing.

Much care should be taken in keeping the stones in good condition. Except in the ways mentioned, no dirt or grit should be allowed to come in contact with their surfaces. A single particle of grit lodged in the surface of the stone will fill the whole surface of the ground section with scratches. Although I keep these stones in a closely fitting drawer, it is necessary to cover each with a cloth that is so closely woven as to exclude all dust.

In taking care of the machine itself, one can not be too careful. All of the bearings of the lathe head and of the grinding machine should be swaddled with candle-wick saturated with oil to prevent the ingress of gritty particles. This is especially needful when using the aluminum saws and feeding them with carborundum powder. Then every bearing about the whole machine should be especially protected to prevent the possibility of getting grit in the bearings. Carelessness in such a matter will quickly ruin a fine bit of mechanism. But with this care, such a machine should continue to do its work well for a lifetime.

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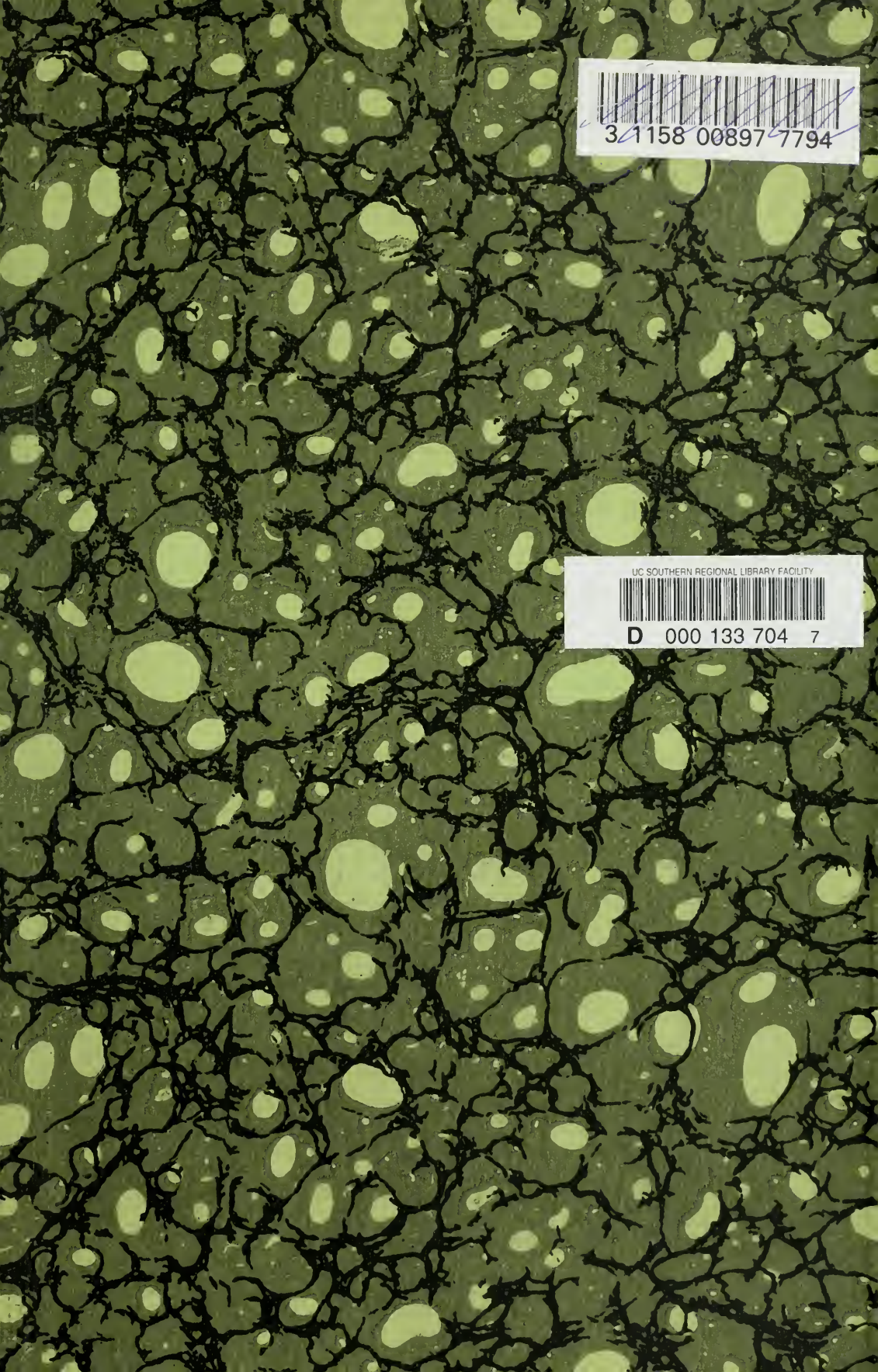
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